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Table of Contents

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ORIGINAL ARTICLES— PAGE.
The Beattie-Smith Lectures—Lecture III: "Some Activities of a Mental Hospital Laboratory During Thirty Years", by OLIVER LATHAM, M.B., Ch.M. .. 797
"Obesity", by E. H. STOKES, M.B., Ch.M. .. 804

REPORTS OF CASES—
"Strychnine Poisoning in a Child", by CHAS. PERRY 812

REVIEWS—
A Text Book on Histology .. 812
Inhalational Therapy .. 813
Psychiatry for the Public .. 813
The Physiology of the Eye .. 813
Embryology .. 814

NOTES ON BOOKS, CURRENT JOURNALS AND NEW APPLIANCES—
Three Short History Books .. 814

LEADING ARTICLES—
Preventive Surgery .. 815

CURRENT COMMENT—
Disposal of Night-Soil .. 816
The Electrocardiographic Diagnosis of Coronary Occlusion .. 817

ABSTRACTS FROM CURRENT MEDICAL LITERATURE— PAGE.
Medicine .. 818

SPECIAL ABSTRACT—
The Anæmias of Infancy and Early Childhood .. 820

BRITISH MEDICAL ASSOCIATION NEWS—
Scientific .. 824

POST-GRADUATE WORK—
Annual Refresher Course in Melbourne .. 826

OBITUARY—
Frank Nyulasy .. 827
Philip Sylvester Clarke .. 828

BOOKS RECEIVED .. 828

DIARY FOR THE MONTH .. 828

MEDICAL APPOINTMENTS VACANT, ETC. .. 828

MEDICAL APPOINTMENTS: IMPORTANT NOTICE 828

EDITORIAL NOTICES .. 828

The Beattie-Smith Lectures.[1]

(UNIVERSITY OF MELBOURNE).

By OLIVER LATHAM, M.B., Ch.M. (Sydney),

Pathologist to the Mental Hospitals Department, New South Wales.

LECTURE III.

SOME ACTIVITIES OF A MENTAL HOSPITAL LABORATORY DURING THIRTY YEARS.

THE PATHOLOGY OF DIVERS MENTAL STATES.

Landry's Paralysis.

FOUR or five clinical cases of Landry's paralysis have been submitted. Two had marginal areas of myelitis as revealed by Marchi's stain, and the other three included two cases wherein the polar cells showed subacute changes with but little surrounding reaction. The fifth turned out to be a case of polioencephalitis, meaning that the cord grey matter showed typical poliomyelitis and this condition could be traced right up to the Betz cells, some of which were quite knocked out.

Anterior Poliomyelitis.

Our laboratory was linked up with the poliomyelitis commission and examined monkeys' brains and cords. We learned much from the examination of the whole nervous system and from noting the extent and location of the lesions. The School of Public Health and Tropical Medicine was testing out the value of immune serum and in one case this did not apparently give protection. However, a careful examination of the monkey's horn cells showed a typical primary or nutritional change or degeneration without any surrounding reaction. This picture[61] is not usually that of poliomyelitis, unless the infection is introduced by injection into

[1] Delivered December 5, 7 and 8, 1933.

the sciatic nerve, whereas this infection was intracerebrally injected and it seemed fair to suggest death from other causes. Also the animal had died sooner than it would have died even if unprotected.

About this time many brains and cords were received from the country; they were from persons dead of poliomyelitis; and in these the involvement of medulla and midbrain suggested *encephalitis lethargica*.

Encephalitis Lethargica.

Perhaps some eighty brains have been submitted to us with a diagnosis of *encephalitis lethargica* and an article has been published detailing the findings. Our ideas of the pathology of this condition as well as of many other intracranial states must be modified in view of the increased importance of the rôles of the microglia and oligodendroglia.[58] One is often asked about the histopathology of the chronic or latent state. Articles have appeared in various scientific papers showing this quite well, especially the astrocytic reaction by the Cajal-Ziehan gold sublimate impregnation. The medulla of a child dying under an anæsthetic being submitted, there was readily shown this same reaction together with perivascular infiltration with mononuclear cells.

The gross hemorrhagic cases[57] with general pink appearance of brain on section are rare with us, so mention must be made of one such wherein gross naked eye hæmorrhages (some a centimetre across) were common in brain stem and cerebellum. There was a history of extensive nasal operations, but no pus cells could be seen and there were other signs of encephalitic reaction.

The Chronic Myelitides.

The term chronic myelitides is used to designate the combined scleroses occurring in conjunction with pernicious anæmia, Addison's disease, diabetes, pellagra, lathyrism or any state wherein the necessary nutritional or toxin neutralizing materials are deficient in the organism.[52] To explain similar lesions in Friedreich's disease it is suggested that here the spinal arteries are congenitally small and inefficient, in a word that insufficient nutriment reaches the cord. Further on I shall show where among other congenital anomalies very small hearts and blood vessels may obtain in imbeciles. Many cords exhibiting posterior and lateral tract demyelination are sent and in a few cases the ventral pyramids too. These cords are often remitted without attempt at diagnosis and sometimes the condition is quite unsuspected. Seldom are any peripheral nerves included; however, the anterior and posterior nerve roots usually are healthy. Sometimes there is great cord atrophy with brittleness and rarely the horn cells are deeply involved.[64] At times patches of demyelination reach the cerebrum with characteristic spongy appearance. Twice the cord lesion clinically indicated a pernicious anæmia, while as yet the blood picture was normal.[65]

The Group Combining Demyelination and Gliosis.

Chronic multiple sclerosis, encephalomyelitis, post-vaccinal myelitis, acute disseminated sclerosis, and certain toxic phenomena associated with food poisoning have not always been classified together; but animal experiments, such as intravenous injections with weak acids, have produced histological pictures typical of all of them. Productive arteritis as in *lues*, gliosis and demyelination, and various round cell reactions, can thus be obtained which serve to link these pictures together, however widely different their ætiology.

The more acute forms are characterized by the following features: around the blood vessels of the subcortical white matter, of the brain stem, of the subependymal tissue, around the periphery of the cord or around its blood vessels are found zones of demyelination with simultaneous destruction of the axis cylinders and a perivascular gliosis mostly of microglia. The more chronic types of disseminated sclerosis, however, show a remarkable persistence of the axis cylinders in the sclerosed areas and these areas may occupy any area in the cord (seen transversely), are not so perivascular, are not anatomical in position, and do not persist for such lengths in the cord as in the encephalomyelitic type. The following case provided some connecting links:

A child aged three years[65] had died with mysterious nervous phenomena, and her brain had revealed widespread and extensive productive arteritis surrounded by areas of the most definite demyelination, softening and in places glial proliferation, cells with abundant long fibres. And the glia was most abundant of all near the hippocampus and posterior end of the lateral ventricles, places called by Weil "*Metterwinkel*", storm centre or pocket.

This is the place where gliosis may predominate or first show up in those curious diseases among which we must note Schilder's disease. Only a few cases of encephalomyelitis have come to hand, one of which I published.[9] Of eight cases of multiple sclerosis, two were of the acute type, both curiously enough sent at 15 years' interval by the same man; thus they are rare, and authors tell us are never diagnosed clinically. This is not surprising, because body symptoms are so acute, resembling acute myelitis. Indeed the first was a peculiar type of transverse myelitis and the second case also included the histopathology of *encephalitis lethargica* in addition to the cord pathology. We often see multiple sclerosis diagnosed as hysteria and perhaps for this reason. I have followed the gliosis right up to the ventricles,[66] and certain writers maintain that an early gliosis affecting the thalami may cause indefinite emotional phenomena long before definite organic signs are patent.

Syphilis of the Nervous System.

General paralysis and tabes[67] need not detain us, beyond noting the changes effected by malarial treatment (Figure XXI). I am not prepared to say that one can always follow the different stages wherein parenchymatous paresis becomes changed into curable gummatous granulomatosis after malaria,[68] including mesenchyme reactions and

endothelial proliferation. From time to time other forms of neural *lues* come along. A case of apparent luetic myelitis with positive serum and fluid reactions *post mortem* turned out to be combined sclerosis. Another type was the cord of a

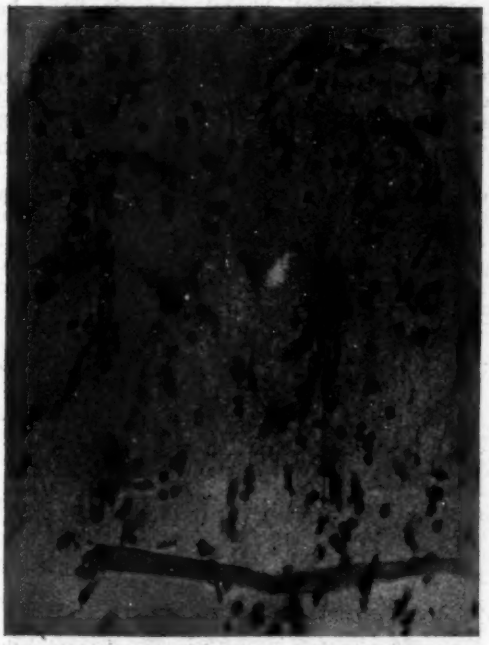

FIGURE XXI.
Rod cells in rather an acute case of paresis or encephalitis. Formalin fixation and methylene blue preparation. × 400.

child of eight years which showed old luetic blood vessels extensive granulomatosis and distortion and dilatation of the central canal, doubtless an example of developmental error due to *lues*. Several brains have been received, the seat of large cavities due to gas organisms. There is no doubt but that the routine Bordet-Wassermann tests reveal many incipient cases of paresis previously exhibited as interesting neurological cases.

Other Infective States.

Elsewhere I have alluded to peculiar brain and cord reactions, in which the lesions are sterile,[69] but in which some other part of the body is infected. Perhaps for these reasons the brains of hospital patients are not all necessarily healthy or samples of the normal. To illustrate this, I want to allude to the following case:

A female patient, aged twenty-nine years, from a general hospital, had neck rigidity and left-sided Babinski reflex, some rigidity of the lower limbs, and mental dulness. Spinal fluid was normal, save for blood. Microscopically there were found innumerable areas of cerebral softening under the pia, with *Gitterzellen*, but no glial reaction. There was some reaction such as mononuclear cells in the pia and a few red cells. The provisional diagnosis of encephalitis could not be gainsaid. Multiple demyelination under pia of both cord and cortex has been described with meningitis, but pus cells were not in evidence here. (Figure XXII.)

Is this a case in which definite brain damage has obtained without signs of brain infection, but where evidence of bodily infection elsewhere was suspected? The patient had had severe headaches, vomiting, and cough for a week. Respirations were forty a minute, with moist râles and rhonchi. Localized swellings of brain tissue may suggest tumour formation, when actually due to infective or to non-infective reaction.[72] [73]

A form of *encephalitis lethargica* has been described accompanied by symptoms of amyotrophic lateral sclerosis and cord involvement.

One such was long a patient at Broughton Hall, thin and emaciated, with certain signs suggestive of brain stem disease, rigidity *et cetera*. Among the lesions noted were a definite marginal myelitis of the cord, somewhat deeper near the crossed pyramids, and some cell loss.

Once an Indian psychotic gave trouble by repeatedly swallowing his tongue. Wearying of the constant calls of the attendant to relieve choking symptoms, I tied it securely, supposing, as he had a raised temperature and slow pulse, that some intracranial infection was present. *Post mortem* examination showed congealed pus in the interpeduncular space.

Syphilis at times causes great thickening and pial infiltration here, and now Gordon Holmes calls attention to cases in which microscopical infiltration by secondary carcinomatous cells[70] may take place

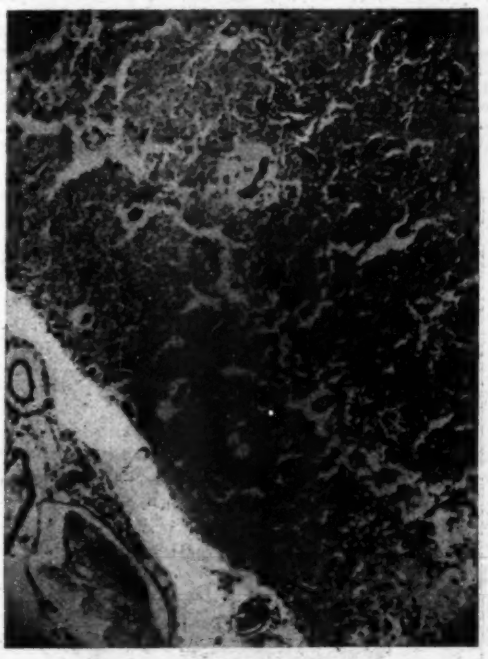

FIGURE XXII.
Disseminated microscopical cortical softenings in *encephalitis lethargica* or associated with septic pneumonia. × 50.

with cranial nerve involvement. Isolated cerebral abscess once caused a wandering sailor to seek an asylum with us; but gross pustular meningitis is rare with us. I have had to go elsewhere for examples of tuberculous meningitis; however, a well-known surgeon rebelled against my diagnosing as a tuberculoma with bacilli *in situ* a nice tumour he had

removed (with excellent clinical results) from a young girl's cerebellum. Professor Wright calls my attention to recent work which might explain the relative rarity of this meningitis. It appears that tuberculous meningitis can eventuate only when tuberculous material is directly injected (or in Nature ruptures) into the subpial space. Sometimes cords have been seen which would not take on good myelin stains, which were soft and friable, with pia loaded with mononuclear cells—some form of acute reaction. The pathological changes of the following three children, from whom specimens were received within a few days of each other, come to me.

In the first case the brain showed such hæmorrhagic encephalitis and stem involvement as to simulate *encephalitis lethargica*, but the cord revealed anterior poliomyelitis of the most florid type I have ever seen.

In the second case similar pathological change to the last was found in the brain, with the brunt round the aqueduct. But, as the reaction increased in evidence as we proceeded down the medulla towards the cord (which was not included), poliomyelitis had to be the diagnosis. The throat was positive for Klebs Löffler bacilli.

The third case was that of a boy who for three weeks had suffered from sharp and shooting pains on walking. All reflexes were normal, no muscular tenderness was present, but some lumbar curvature was noted. Eight days after admission to hospital paralysis of limbs and bladder occurred, with complete sensory loss from the ninth thoracic vertebra down. The straw-coloured spinal fluid had an excess of globulin and a few red cells and was sterile on culture. The *post mortem* examination revealed purulent exudate on the spinal dura, without bone involvement, and there was pus in the kidney. Section of the mass round the cord suggested a chronic granuloma, but no pus cells or bacilli were seen. (Figure XXIII.) The clinical diagnosis was anterior poliomyelitis or tumour of the cord. The serum and fluid gave no Wassermann reaction, else *lues* would have come to mind.

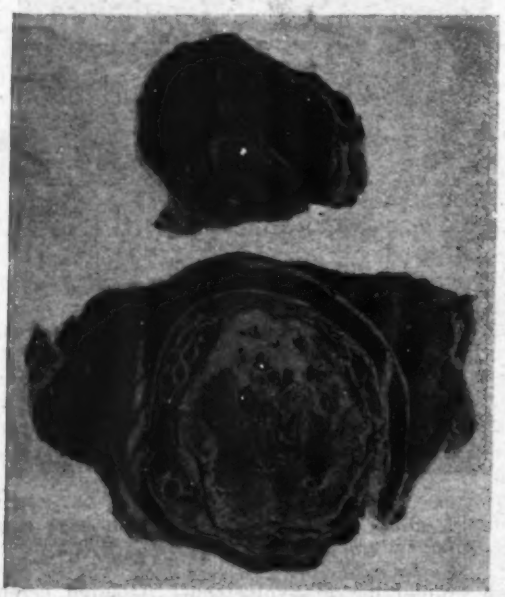

FIGURE XXIII.

Some form of granulomatous reaction round spinal cord of young boy who had *post mortem* septic kidneys. × 3.

I have had similar masses round the *cauda equina* and, with other pathologists, have had difficulty in distinguishing probable granuloma from, say, a meningioma or blastoma.

Motor Neurone Disease.

Under the caption of motor neurone disease no doubt diverse states have been described. Five or six cases of primary lateral sclerosis have been examined, revealing the typical upper neurone affection, the demyelination of the crossed pyramids. Although spastic states are readily recognized, these were not diagnosed till sections were cut. One I must admit deciding was not a lesion until I had sectioned seventeen levels, so slight was the paling; yet the clinical symptoms were marked.

Conditions called progressive muscular atrophy have not been mailed us, but a dozen have in which the motor nerves were thought to be involved.

In five of these the anterior horn cells were so obviously involved that I have included slides of them. (Figure XXIV.)

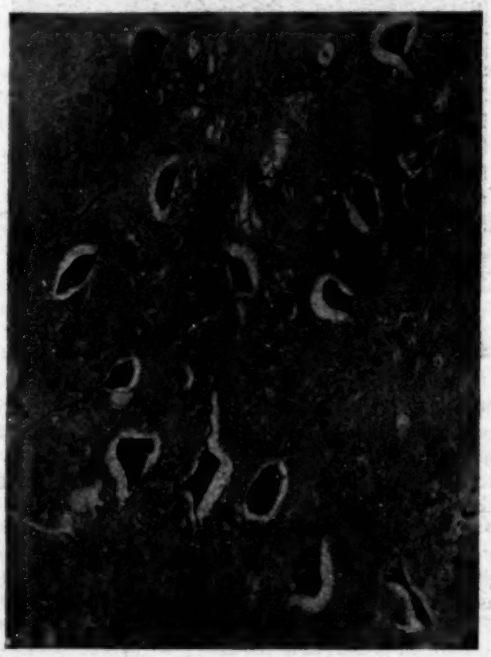

FIGURE XXIV.

Anterior horn cells, showing primary degeneration of the neurone. Cells show all stages from the normal to the ghost cell. × 150.

In one case the illness, which included lower limb paralysis, fever and malaise and general wasting, so affected this sthenic patient's character that he became docile, religious and industrious, till death eighteen months later revealed in both lumbar and cervical enlargements numerous ghost cells, neurones present in form and size, but blessed with no contents.

Beyond collections of nuclei or lymphocytes and interstitial hyperplasia, such as gliosis of the cord and proliferation of cells and fibres in the neural coverings, I failed to find a pathology for *myasthenia gravis* which, after all, can improve in pregnancy. The second case only included brain and cord, and I found nothing.

Von Recklinghausen described a disease in which lumps occurred all over the body, commonly, of course, on the skin, and up to thousands in number. On nerves they are like fibrous tissue, but the histology I will discuss under Schwannomata. (Figure XXV.)

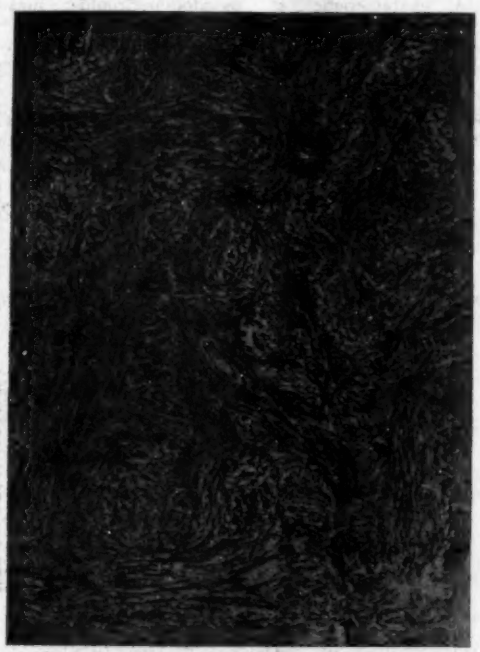

FIGURE XXV.

Schwannomatous tissue from large tumour base of child's brain. Note palisading of elongated nuclei. × 50.

On the dura they are like dura; in the brain they are like glia; in the gut or spinal cord they are like the neighbouring tissue.

A curious variation was one wherein the minute hair-like cutaneous nerves about the knee joint had proliferated till they measured 0·7 centimetre across. Histologically the dozen or so of myelinated fibres had been separated widely apart by Schwann cells, as in the true larger lesions on nerves, where the fibrous tumour is pierced by nerve fibres. In the true single tumour of the Schwannoma group the nerve, if seen, lies on the outside. The nerves regressed as the man's health improved.

Developmental Anomalies.

It might have been supposed that our museum would have held a large and varied assortment of ament's brains. This is not so, because very few necropsies of them are obtained by us. In Victoria and other States the reverse holds good. Nevertheless, we have gained access to a series sufficiently instructive to bring before you. I want first to recall an article on a cyclopic pig described by us in 1924,[74] because the entire absence of both cerebral hemispheres in it and the simple way that the cerebral vesicles (as much as we can see of them) lie quite externally, give the key to some human specimens I am about to describe. In that article Winkler supposes a diseased state of these self-same vesicles to have been the cause of both the cyclops and the non-further differentiation of the vesicles, some anterior fusing, for instance. Other authorities[75] agree with the ependymitis theory, but Sachs and Hausman warn us that surprising retrogressions can occur even after birth, and to use care in ascribing all cystic hemispheres to developmental faults. It so happens there was an endocrine factor in this pig's dam. A diagram and photograph by Professor Berry[76] of an ament shows a very small left cerebral hemisphere in which, as in other parts, the primitive ependymal-lined vesicle shows out on the surface.

A more extreme case is represented by one kindly referred to me by Dr. F. Tidswell from the Royal Alexandra Hospital for Children. (Figure XXVI.)

FIGURE XXVI.

Right view of child's brain wherein the cerebral hemispheres largely remained the primitive vesicles, only the tip of the occiput developing properly. (Cotton wool taking the place of the fluid originally there.) Half normal size.

Except for portions of the occipital and temporal lobes, both hemispheres were simply huge cysts. The offspring of a healthy mother, the infant appeared normal till four months old, when its head began to swell. Ultimately she was admitted to hospital with the diagnosis of hydrocephalus and a cranial measurement of 52·5 centimetres (21 inches). The health remained good and the habits normal till the child was twenty months old, when there developed increasing loss of rectal and bladder control. The child slept normally and was bright when awake; she could see well and take notice, but did not recognize persons. No obvious defect of smell was observed, and she could laugh and smile, and probably felt pain. She never walked, and her legs became contracted at twenty months; they were considered spastic on admission to hospital. She could not talk, but made "little noises" when hungry. She was moribund on admission, and no proper neurological examination was possible. Death was due to bronchopneumonia at three years of age.

Professor Burkitt, who briefly examined the specimen with me, suggests the following notes. The right half of the child's brain and brain stem is available for examination. The optic tract and the postero-lateral geniculate body are probably intact, as are also probably the thalamus and head and body of the caudate nucleus. The whole of the cerebral hemisphere is cystic, except the medial and inner point and tip of the temporal lobe, and the under surface of the occipital lobe, together with a small portion of the inferior temporal convolution externally.

The hippocampus and incus are in part intact. The fornix, *corpus callosum* and *septum pellucidum* seem to be included in the cystic transformation. The anterior part of the calcarine fissure seems to form one of the limbs of attachment of the cyst wall. It is doubtful whether the insular cortex or lenticular nucleus is present. Apparently the *pes hippocampi* can be seen spread out on the floor of the cystic cavity. The amygdaloid nucleus is probably present. The cerebellum, pons and medulla likewise appear normal, but the optic thalamus seems somewhat congested and swollen. It is reported that the other half reveals precisely similar phenomena.

Another case, this time from the Newcastle Mental Hospital (idiots), reveals almost the same condition but in only one hemisphere. Here there was contralateral atrophy of the opposite cerebellar lobe.[77] Dr. Nowland, who remits the following notes, did not think of packing the cyst under water or formalin while still soft, and so it appears shrunken on to the brain stem. (Figure XXVII.)

The child lived to three years, was 90 centimetres (three feet) high, 20·7 kilograms (three stone four pounds) in weight, and had a cranial measurement of 45·6 centimetres (eighteen and a quarter inches). He was very fat, and his right pupil reacted to light, while his left did not. We have good Weigert-Pal preparations of the brain stem and cord of this case, revealing obvious defect in the pyramidal system.

Another patient,[78] with the one hemisphere small and the contralateral cerebellar hemisphere similarly affected, apparently achieved some mental development and did

FIGURE XXVII.

Same type of brain, exhibiting the condition on one side only. There was contralateral cerebellar atrophy. Half normal size.

well at school. Fits at eighteen months ushered in some paralysis of the left side. However, she walked at two, and talked at three and, living to an age of twenty years, she suddenly collapsed and died. The *post mortem* examination revealed megalocolon, a small aorta, with left limbs smaller than the right.

Quite another type is revealed in the next case, which exhibits microgyria, great internal hydrocephalus, hydromyelia and very small cord,[79] the cervical levels of which are flattened. (Figure XXVIII.)

Lastly, there remains a case of an ament of forty odd years whose photographs reveal intense emaciation, with obvious muscular atrophy. Her brain is small and infantile, her spinal cord, as in the others, is extremely small, her heart is 6·75 centimetres (two and a half inches) long, with atheromatous vessels, and her ovaries are eighteen millimetres long and four millimetres thick. Her muscles show a moderate degree of the changes characteristic of pseudohypertrophic muscular paralysis.[80]

FIGURE XXVIII.

Brain of idiot, showing microgyria and extensive dilatation of the ventricles (congenital). Half normal size.

While on this subject, I recall another case.

A neurologist suspected something like this amiss with a boy, and he actually removed muscle from each leg. His chagrin was complete when a pathologist reported them to be normal. He posted them to the laboratory, where special preparations showed great proliferation of the intrafibrillary nuclei.

The following peculiar case merits reporting.

Once I was handed a man's thigh and leg which were as hard as cartilage.[81] It was the second thigh he had had surgically removed for excruciating pain by some surgeon who kindly supplied notes, indicating that the patient had also lost a child[82] from pseudohypertrophic muscular paralysis. Microphotographs of sections of muscle from this amputated leg showed a histopathology like that of his child. On naked eye examination most of the muscles were pink or pinky yellow, degenerated into tendon almost, œdematous, like fibrous tissue to cut, and apparently densely attached to a hard and fibrous epidermis. This man was alive, well and free from pain several decades later.[83] [84]

Now Horn and Aurecchio describe such a limb with hard subcutaneous tissue extending right down to the muscle, and the muscle was similarly affected. They maintain that these characteristics belong to the domain of congenital phenomena. Hospitals for the reception of idiots abound in cases of unilateral spastic paralysis commonly accompanied by epilepsy.

A finely proportioned woman of thirty-five, thus afflicted, died in a fit, and her brain revealed in the opposite rolandic area a sort of contraction or fusing together of the gyri about the middle of the fissure. It looked yellowing grey, and on section appeared pigmented, greatly atrophied and with innumerable neurones destroyed, lost, or hopelessly degenerated. I take it that this was due to true birth palsy after trauma at birth.

Unfortunately I cannot recollect receiving an example of Little's disease,[86] which, while including many clinical types and pathological pictures, yet has for its ætiology a real congenital defect in the power of normal development, and this appellation will thus include types developing illness after an apparently healthy start in life.[87]

Now C. Todde mentions the condition crossed hemiatrophy. He considers it to be rare, and holds that the patients are often epileptics. Sometimes imbecility is more apparent histologically, for example, the finding of 80% of Purkinje's cells in the cerebellum, missing in an ament with four insane sisters.

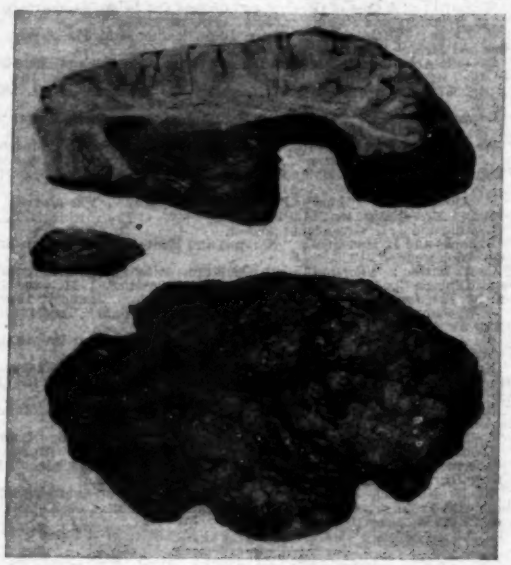

FIGURE XXIX.
Case of epiloia, showing candle guttering of the optic thalamus and central nuclei, and gross tumour formation in kidney. One-third natural size.

Useful articles on epiloia have been written by the late Dr. Lind. We have records of but two cases.[88] (Figure XXIX.)

One of them reveals moderate candle guttering in the ventricles and, microscopically, areas of sclerosis in cortex beneath the pia, the sclerotic tissue replacing many neurones. The kidneys were necrotic and the seat of new growths, the histology of which was not clear, while a section of one of Dr. Lind's cases seemed typical Schwannomatous tissue.

The "new growth tissue" seems to vary in type according to situation, that is, candle gutterings are neuroglia. In arteriosclerosis also can be found sclerosed areas *minus* neurones.

Developmental Abnormalities without Symptoms.

Many developmental abnormalities of slight degree can obtain in the central nervous system, as elsewhere, but the presence of double cords, two instances of which we have, merits a few words.

The first from a severe case of locomotor ataxia was in the form of a heterotopia or projection from the motor area of the cord forwards, and included both grey and white matter. As the lesion was posterior, the heterotopia was healthy save for infiltrated pia. As is usual, many other levels showed disturbances of a kind definitely developmental.

The second was more remarkable, as the cord was double from the second cervical almost to the sacral level, and some extra nervous tissue found its way into the *cauda equina*. (Figure XXX.) One cord surrounded the other, and the end came because the central, and probably the real, functioning one had its circulation interfered with and presented a more decomposed state than its fellow, which included all the elements of grey and white matter. The case was correctly diagnosed as myelitis.

The diagnosis of developmental anomaly is not often received kindly by neurologists, we often find, but why?

FIGURE XXX.
Case showing double spinal cord stained by Weigert-Pal, revealing each portion containing both grey and white matter. ×3.

Pathological Nerves.

Sometimes pathological nerves show a fine fibrosis, sometimes a local excess of scar tissue; at times they are the seat of some Schwannomatous proliferation or of von Recklinghausen's disease, and surprisingly often of some form of hæmorrhage. Hæmorrhage about the *cauda equina* and chronic engorgement in its vicinity can cause severe neuralgia. At other times myelin degeneration can be detected, or actual destruction of nerve fibres.

Pink Disease.

Some eight or nine cases of pink disease have been submitted. Except in one case which showed excessive nuclei in the anterior horn and about the polar cells, I failed to note any characteristic pathological change. Once I fancied that definite paling of the crossed pyramidal tract area connoted a form of nutritional myelitis (and American authors, too, have commented on this), but the examination of numerous controls of under one year old norms and the writings of Lucas and Hewer[89] convince me that the demyelination was a developmental phenomenon. I join issue with Wilfred Evans in holding that "pink" phenomena, like jaundice, are but a sign of some unknown incorrect vital function.

THE DIPLOMA COURSE IN PSYCHOLOGICAL MEDICINE.

In the diploma course in psychological medicine at the University of Sydney, oral and/or practical lectures are given in psychology, neurology, anatomy, physiology, psychiatry and neuro-pathology.

The lectures in pathology aim at enabling medical officers to gather in pathological material correctly, to perform simple, but important, tests, and to understand better the nature of nervous and mental disease.

Post mortem examinations are performed at mental hospitals, the students thus being enabled to obtain good specimens, to preserve them, and to pack them suitably for transmission to the laboratory. The senior resident medical officer sees to it that they also gather correctly specimens of blood, spinal fluid, urine, fæces, swabs *et cetera*, and correctly preserve them for early transmission.

A set of lectures is given on the pathology of every-day nervous and mental diseases; these are always accompanied by illustrations. The illustrations are provided by epidiascope demonstration from book or monograph, lantern slides, or by microscopic slides. The clinical history and anatomical basis are appealed to where possible, and association is kept up with the professors of medicine and psychiatry.

Museum specimens from mental hospitals and other sources are available to illustrate any points.

Boxes containing slides of neurological interest, and often alluded to in the above lectures, are provided. These are specially mordanted and stained to bring out neuro-pathological points. Often type-written books accompany these boxes, so that they can be studied at leisure. Practically a library of slides is available wherein most nervous diseases can be examined.

Typewritten or printed leaflets are provided for the spinal fluid, and many of the manipulations and tests are performed here.

In the laboratory the students fix, mordant, cut, stain and mount sections, whether frozen, paraffin or celloidin, and prepare slides and cultures from other specimens. Biochemical tests are done, namely, blood urea, sugar, uric acid *et cetera*, estimations and the Kline and Wassermann reactions are noted. Urine from malarial patients and blood slides are specially emphasized. Agglutination tests for the typho-colon group are readily performed with formalinized cultures by the laboratory "bedside" method. Museum specimens are mounted, and dark ground methods with the microscope are practised. For nearly fifteen years this laboratory annually demonstrated dark ground methods and the use of the microscope to the fourth year students. Lastly, many Weigert-Pal lesions reveal anatomical tracts, and are constantly being used by our university lecturers in anatomy and medicine.

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OBESITY.[1]

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FIRST of all I wish to thank the North Shore Clinical Society for the honour that it has done me by its invitation to address the members tonight.

The subject chosen for discussion is obesity, a commonplace condition which, in spite of the fact that its causation is obscure, can in many instances be benefited by suitable therapeutic measures. While loss of weight is always regarded seriously both by the laity and the medical profession, an increase in weight is often considered to be an indication of robust health and therefore not worthy of notice. Life insurance companies, however, do not look upon overweight subjects with favour; for extensive observations have shown that the mortality rate among the obese is considerably higher than among

[1] Read at a meeting of the North Shore Clinical Society on October 3, 1933.

people of normal weight. Thus in an investigation of 744,672 policy-holders of thirty of the larger American companies[1] it was found that there was no increase in the mortality rate among persons who were up to 4·5 kilograms (ten pounds) overweight, but above that figure the rate rose steadily and alarmingly.

Obesity may be defined as a condition in which there is an excessive deposit of fat throughout the body; but, as has been pointed out by C. Bickerton Blackburn,[2] it is difficult to supply a complete and satisfactory definition. The condition of "overweight" gradually merges into obesity, and the point where one ends and the other begins is often difficult to determine. Assistance may be obtained from standard tables of heights and weights, such as that compiled by the Association of Life Insurance Directors and the Actuarial Society of America.[3]

It is customary to describe two varieties of obesity.

(1) Exogenous obesity, in which there is a faulty balance between the intake of food and the output of energy.

(2) Endogenous obesity, which may result from endocrine or metabolic disturbance.

In practice, however, it is found that while there are clearly defined instances either of exogenous or endogenous obesity, the majority of cases of obesity present features common to both varieties.

It is proposed in this paper to discuss the ætiology, pathogenesis, clinical features, diagnosis, complications, prognosis and treatment of obesity.

DATA.

During the past five and a half years a number of people suffering from obesity have come under my observation, and it is proposed to illustrate this paper by reference to these patients. Fifty cases have been selected for this purpose. They may be classified as follows:

1. Generalized obesity (including exogenous obesity)	24 cases
2. Obesity of pituitary type	15 cases
3. Obesity associated with myxœdema	6 cases
4. Obesity apparently following oophorectomy	3 cases
5. Obesity associated with endocrine disturbance of ill-defined type	2 cases

ÆTIOLOGY.

The following factors are of importance in the causation of obesity: (i) heredity, (ii) sex, (iii) temperament, (iv) occupation and enforced rest, (v) diet and exercise, (vi) age, and (vii) endocrine disorders.

Heredity.—Obesity is frequently traceable through several generations. James M. Anders[4] found 300 instances in 543 cases. Hereditary predisposition usually manifests itself early in life; but it may not show itself until a later period or until some exciting cause becomes operative. Many instances were noted in the cases under review of corpulent parents with obese children, and one example of a brother and sister suffering from obesity is given.

Sex.—Obesity is much commoner in the female than in the male. In the series of patients studied, 37 were females, while only 13 were males. Though due importance must be placed upon the more quiet and less active life led by some women, and their greater tendency to indulge in fat-forming articles of diet, none can deny the association of puberty, pregnancy and the menopause, natural or artificial, with obesity. Two of the patients studied noted the onset of their obesity in the puerperium.

Temperament.—The indolent, sluggish, luxury-loving and rest-loving individual is predisposed to corpulence. Thus obesity is commonly observed among the Dutch and certain Oriental and Mediterranean races. A nervous, restless individual expends more energy than a placid person in carrying on certain definite work, and is less apt to become obese.

Occupation and Enforced Rest.—Sedentary life favours the development of corpulence. Enforced rest following accidents or acute illnesses is often the starting point of obesity. Thus one of the patients studied had increased 12·6 kilograms (two stone) in weight after an attack of pneumonia. The influence of rest after gynæcological operations and after the puerperium must be taken into account in assessing the true importance of ovarian dysfunction in the causation of obesity.

Diet and Exercise.—Exogenous or simple obesity is dependent upon the ingestion of abnormally large amounts of fat-forming foods with or without deficient exercise. It is apparently the foods of a starchy nature, rich in carbohydrates, such as bread and potatoes, which are largely responsible for this form of obesity. Intemperance in the use of alcohol is also an important factor; for alcohol is readily oxidized and thus allows the fat already formed to remain undisturbed. Further, certain alcoholic beverages, such as beer, contain a certain amount of carbohydrate. Alcohol played a part in the causation of obesity in two of the cases studied. With regard to exercise, it is frequently found that a man who has indulged in strenuous athletics in his youth becomes obese upon retirement from sport.

Age.—Excepting when the endocrine glands are involved obesity usually arises in the female between the ages of thirty and fifty years, and in the male between forty and fifty. Infants, especially those fed on artificial foods, frequently become exceedingly obese.

Endocrine Disorders.—Disorders of the thyreoid, pituitary, adrenal and reproductive glands may play a part in the causation of obesity. The special characters of endocrine obesity will be discussed later.

PATHOGENESIS.

A discussion of the various theories relating to the origin of obesity will be of interest and value.

While it is possible to attribute corpulence in some persons to dietetic excesses, it is well known

that many obese people eat remarkably little. Strouse, Wang and Dye[5] noted that obesity might occur in certain persons on intakes much below their calculated caloric requirements. Although it is evident that the storage mechanism in such people is excessively well developed, the origin of the fat accumulations is a subject that is imperfectly understood.

E. P. Poulton,[6] in his presidential address to the Section of Therapeutics and Pharmacology of the Royal Society of Medicine, stated:

> It has often been considered remarkable that most people should keep the same weight from year to year, which means that the amount of food taken in is exactly balanced by the amount burnt in the body.

He went on to say that there was general agreement as to the three processes responsible for the burning, namely: (i) the so-called basal metabolism of the individual, (ii) the specific dynamic action of foodstuffs, and (iii) exercise. Furthermore, he emphasized the fact that although in many obese people the basal metabolic rate was low, yet related to body surface it might be normal. In such cases hypothyreoidism has been diagnosed; but this is not necessarily the case, for the activity of glandular and muscular tissues may be up to normal standard. In such instances it would be reasonable to assume that, if the basal metabolic rate were normal, a state of hyperthyreoidism might be present. On this theory, hypothyreoidism may follow obesity, in the same way as patients suffering from Graves's disease may eventually become myxœdematous. The importance of this observation in treatment is obvious; for thyreoid substance cannot be expected to produce a beneficial increase in the metabolism in the cases with a normal basal metabolic rate. But when the metabolism is low, thyreoid substance may be given, with marked improvement in the symptoms, but generally with very little loss of weight, unless dietetic measures are also taken; for the feeling of *bien être* engendered by the treatment stimulates the patient's appetite. It is of importance to note that Strouse, Wang and Dye[5] found that there was no characteristic change in the basal metabolism of the obese, and furthermore they showed that the specific dynamic action of protein was decidedly less in the obese than in the normal person.

Poulton[6] also discussed the hyperinsulinism theory of obesity, first suggested by Falta, and stated that the following were reasons in its favour:

> (*a*) Obesity not uncommonly overtakes diabetic patients who are treated with insulin.
>
> (*b*) Insulin and carbohydrate have been used in fattening lean people.
>
> (*c*) The frequency with which diabetes occurs in obesity is compatible with the wearing out of an over-active function.
>
> (*d*) The sugar tolerance of obesity is increased in some cases, which is compatible with an increased output of insulin.
>
> (*e*) It has been shown by many observers that repeated ingestion or injection of glucose stimulates the pancreas to secrete insulin, as indicated by the fact that the blood sugar does not rise so high after the second administration.

Against this theory it may be stated that case reports of patients suffering from hyperinsulinism as a result of tumours of the islets of Langerhans show that obesity was not a pronounced feature.

The suggestion that the exogenous type of obesity may be the result of pancreatic stimulation following the ingestion of too much carbohydrate is attractive; for this stimulation may well lead to some hypertrophy of the islet tissue which will cause the blood sugar content to fall and produce feelings of hunger which must be satisfied by more food. Thus a vicious circle may be established.

The part played by the endocrine glands in the causation of obesity is important and interesting. The pituitary gland, which Langdon Brown[7] has called "the leader in the endocrine orchestra", plays an important rôle in the production of some forms of obesity; but the relationship of the pituitary gland to obesity is complicated by the fact that disease of the hypothalamic region of the brain may produce identically the same changes as lesions of the gland itself. In three of the cases studied it is possible that the hypothalamic region, rather than the pituitary gland, was involved. Furthermore, there is a close relationship between the pituitary gland and the thyreoid gland and the gonads. Lambie[8] has recently described the various syndromes resulting from the interaction of the gonadotropic, thyreotropic and growth factors of the anterior portion of the pituitary gland.

Clinical Features.

While the dictates of fashion cause some patients to seek medical advice for cosmetic reasons, the majority of sufferers from obesity usually consult their doctors on account of the discomfort produced by the pressure of large masses of superfluous adipose tissue. In the series under consideration, breathlessness on exertion, palpitation, and swelling of the legs and feet were the most common presenting symptoms. The dyspnœa was caused in some cases by the excessive amount of fat hampering the action of the heart; in others by actual cardiac disease. The blood pressure was definitely raised in 14 out of 36 cases in which it was recorded. It is of interest to note that Aubertin and Coursier[9] rejected completely the assumption that obesity in itself could cause high blood pressure. But the frequent association of obesity with morbid conditions in which the blood pressure is high should be remembered.

Menstrual irregularity was frequently present. Pain in the region of the knees, or actual arthritis accentuated by the increasing weight, was noted in five cases. Albuminuria was present in some cases.

Although most of the patients studied were of the plethoric type, anæmia is frequently found in patients suffering from obesity. Its importance lies in the fact that the diminished oxygen-carrying capacity of the blood increases the sluggishness induced by the obesity.

In spite of the commonly accepted idea that fat people are endowed with a pleasant type of mentality, it was found that a number of the people observed were of an irritable temperament. Perhaps had the immortal bard's Julius Cæsar to deal with

some of the modern patients suffering from obesity he would not have said: "Let me have men about me that are fat."

FIGURE Ia.

FIGURE Ib.

Generalized obesity (Case X), showing umbilical hernia.

Physical Characteristics of Obesity in Endocrine Disturbance.

Although Strouse, Wang and Dye[5] state that "some of the anatomic localisations of fat deposits that have been described as pathognomonic of disturbances of special glands are of more interest from the standpoint of clinical speculation than of proved fact", yet the localization of fat deposits in certain areas of the body is considered by many authors to be of great importance in the diagnosis of endocrine disorders.

Engelbach,[10] writing in 1922, stated that obesity was one of the most important diagnostic signs of ductless gland disturbance. In his opinion the following two points were of great importance: (i) the age at which the obesity occurred, (ii) the localization and distribution of the obesity.

The Age Incidence of Obesity.

Engelbach grouped the obesities according to their age incidence into three divisions: (*a*) infantile, (*b*) juvenile, (*c*) adult.

Infantile Obesity.—Engelbach stressed the fact that, if unexplained excess weight at birth be followed by late eruption of the first teeth, delayed locomotion and retarded speech, it should be suspected that cretinism is present, and a therapeutic trial of thyreoid substance should be made. As a rule, these hypothyreoid babies do not carry their excessive weight into early childhood.

Juvenile Obesity.—Engelbach attributed the juvenile obesities to disorder of the pituitary gland. As a rule, development is normal up to about five years of age; but between this age and adolescence these patients suddenly become obese. In contrast to that of the hypothyreoid child, their mental development is not retarded, and in fact they are frequently precocious.

Adult Obesity.—The obesities commencing in adult life may be due to disorder of the pituitary or thyreoid gland or the gonads. Engelbach stated that with the exception of adiposity localized over the trochanter region, obesity due to gonadal insufficiency rarely occurred before the age of thirty-five. Thus we see that in obesity occurring before the age of thirty-five, the pituitary and the thyreoid glands may be at fault, either separately or singly. He stated that the later the obesity occurred, and the longer its duration, the greater the probability that more than one gland was involved, and the worse the prognosis.

Localization and Distribution of Obesity.

Obesity associated with Thyreoid Deficiency.—The clinical picture of myxœdema is so well known that there is no need to elaborate it. The enlargement of the hands, the supraclavicular pads, and the evenly distributed "solid œdema" are characteristic; but other classical features, such as the coarse facies, the thick everted lips, the puffy eyelids, the malar flush, the wasting of the eyebrows,

FIGURE II.

Obesity associated with myxœdema (Dr. Ritchie's patient).

the dry skin, and the brittle hair, will immediately point to the diagnosis. Although sufferers from myxœdema are frequently stout (Figure II) in many instances, as has been previously pointed out,[11] they are of a thin build. As a matter of fact, the increase in weight found in myxœdema is

due to an accumulation of water, albumin and salt in the tissues rather than to adipose tissue.

Obesity due to Pituitary Gland Disturbance.—Obesity due to pituitary gland disturbance is localized about the hip and shoulder girdles (Figures IIIA and IIIB). The pelvic girdle obesity extends from the umbilicus down to the junction of the middle and lower thirds of the thigh. The shoulder girdle is less noticeably affected as a rule, the deltoid region being especially prominent. The limbs are usually slender and gracile. Besides these localized areas, there is, as a rule, considerable increase of fat in the lower and lateral parts of the thorax. The mammary region is often affected, and an abdominal apron of fat is usually present.

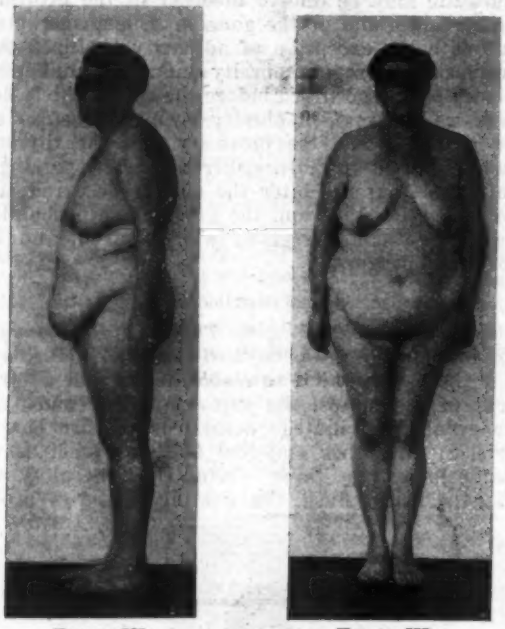

FIGURE IIIA. FIGURE IIIB.
Obesity of the pituitary type (Case VI).

FIGURE IV.
Obesity of the pituitary type (Case IV).

Obesity due to Gonad Insufficiency.—Engelbach stated that in gonad insufficiency the most striking feature was the complete absence of obesity, with the exception of that round the trochanteric region, until the late age of thirty-five. The typical eunuchoid appearance (Figures VA and VB) seen in young people is characterized by an overgrowth of the long bones and a disproportion between the upper (short) and the lower (long) measurement, with an absence of obesity, except in the trochanter region. Engelbach described true gonad obesity as consisting of disproportionate mammary enlargement, as compared with the obesity in other parts of the body, with addition of an area of *mons veneris* shelving and a decided localized trochanter padding, with a diffuse, even fullness of the thighs, extending to the knees. It is sometimes difficult to distinguish clinically the types of obesity due to pituitary and gonad disorder, and in view of the recent work on the pituitary gland, to which reference has already been made, the reason seems obvious.

FIGURE VA.
Eunuchoidism.

FIGURE VB.
Eunuchoidism.

Pluriglandular Obesity.—Defects of more than one gland may give rise to the so-called pluriglandular obesity. The commonest example is a

combination of pituitary and thyreoid obesity. Rare conditions, such as Dercum's disease (*adiposis dolorosa*), and the Lawrence-Biedl syndrome (adiposity of the pituitary type associated with *retinitis pigmentosa*, mental deficiency and polydactylism), are worthy of mention. That adrenal disorder (for example, adrenal virilism), an example of which was described by A. W. Holmes à Court,[12] may be the cause of obesity should be borne in mind.

Diagnosis.

The diagnosis of well marked obesity should present no difficulty. An increase in weight due to œdema, such as is found in myocardial failure, should not be mistaken for obesity. As has been stated in the discussion of obesity due to glandular deficiency, the age incidence and the distribution of the obesity are important factors. The longer the duration of the endocrine disorder, the more likelihood there is of secondary glandular participation.

Complications and Sequelæ.

Among the principal complications are those referable to the circulatory system, such as anginal attacks, arteriosclerosis, coronary thrombosis and cerebral hæmorrhage. Bronchitis and pulmonary congestion are common. Arthritis of the knee joints, due to increase of weight, was often seen in the series of cases under consideration. Intertrigo was also a common and unpleasant complication. Joslin and others have stressed the occurrence of *diabetes mellitus* in overweight persons. Cholecystitis and gall-stones are frequent in the obese.

Gout, apparently not a common disease in Australia, was noted in one of the series. Hernia was also seen in two instances. It is a well known clinical observation that obese subjects react badly to acute illnesses and are bad operative risks, owing to their large bulk rendering surgical access difficult, and to their liability to post-operative pneumonia and other complications.

Prognosis.

Prognosis depends first upon the degree of obesity, and secondly upon the complications. In the earlier stages, where dietetic excess is the main feature, the prognosis is quite good. In more advanced cases, with involvement of the circulatory system, the prognosis is usually very grave, depending, as it does, upon the vascular condition. With regard to the cases in which the endocrine glands are involved, the patients whose "obesity" is due to myxœdema respond well to treatment, but, as has often been seen, do not necessarily lose much weight. The prognosis in the cases due to pituitary and gonad involvement is much less favourable.

As has already been mentioned, life insurance companies take an unfavourable view of obese persons. Where an obese person is addicted to over-indulgence in alcohol, the gravity of the prognosis is increased.

Treatment.

Prophylaxis is essential, and children of obese parents should avoid farinaceous foods and take a moderate amount of exercise. Unfortunately it is found that these children are trained by their parents in bad dietetic habits. In middle life all imprudences in eating and drinking should be avoided.

Before treatment is instituted, the patient should be carefully examined, special attention being paid to the circulatory system; and the weight and height should be recorded. It is as well to remember that in certain cases it may not be wise to attempt to reduce weight. Treatment may be conveniently discussed under three headings: "diet", "exercise" and "glandular therapy".

Diet.

The chief points are to reduce the total calorific value of the food by the restriction of articles of diet rich in carbohydrate and the reduction of the amount of fat taken to a minimum. The carbohydrate is taken chiefly in the form of green vegetables. Bulk can thus be obtained and the appetite satisfied. A low fluid intake is neither necessary nor desirable, except in cases with cardiac complications. Obesity can be safely reduced on a great variety of diets so long as sufficient protein and carbohydrate are given to prevent the loss of body proteins. In the older dietetic systems, one of the best known of which was that devised by Banting, the proportion of protein was increased while the carbohydrates and fats were reduced. In addition, Banting allowed wines and spirituous liquors, a concession which no doubt added to the popularity of his *régime*.

Admission to hospital, the careful calculation of the caloric value of the food intake, and the weighing of all articles of diet may be necessary in some cases.

A satisfactory diet should contain a sufficient amount of protein in the form of lean meat and fish, a moderate amount of carbohydrate, chiefly in the form of vegetables, and little fat. Of fish, salmon, herrings and sardines, which contain more than 10% fat, should be avoided. Poultry, with the exception of duck (30% fat) may be allowed. Vegetables, such as potatoes, which contain more than 10% carbohydrate, should be used very sparingly. Sugar, bread, cakes and sweets should be almost eliminated. One or two thin slices of bread (which contains 50% to 60% carbohydrate) are the maximum that can be allowed. This applies to all forms of bread. Fresh fruit may be allowed in moderation; but, as in the case of vegetables, fruits containing more than 10% carbohydrate should be restricted. A sufficiency of vitamins is essential. Fried foods should be avoided. Alcohol should be forbidden.

As H. Gardiner-Hill[13] has said, it should be rarely necessary to calculate the caloric value of the food intake; but the prescription of a diet of

known value is sometimes more helpful than general directions. D. M. Dunlop and R. M. Murray Lyon,[14] in the treatment of 523 patients with obesity, used a diet containing 100 grammes of carbohydrate, 66 grammes of protein and 38 grammes of fat, yielding 1,006 calories.

It is important to control the body weight by weekly measurements. In most cases it is best to arrange the diet so that the weight is reduced gradually by one to two pounds a week. This prolongs the period of dieting and accustoms the patient to a small amount of food, so that he is less likely to overeat again.

Exercise.

Exercise, which should be graduated, is best obtained by walking out of doors. People who are able to play such games as tennis and golf should pursue their favourite sport with moderation, provided there is no circulatory contraindication. Gymnastics and special exercises may be advisable in certain cases. When the degree of obesity prevents active exercise, the chief substitutes employed are massage, active and passive movement, and faradization in the form of the Bergonie system, by means of which the principal muscles are made to contract in regular order against a load. Balneotherapy, consisting of hot and cold baths, followed by active hand rubbing, may be a useful adjunct to exercise. The treatment adopted at various well known European watering places, consisting of diet, exercise and the administration of medicinal waters, is specially suitable to the plethoric types of obesity.

Glandular Therapy.

The common practice of using thyreoid substance only in the treatment of obesity cannot be too strongly condemned. Those people whose basal metabolic rate is below normal will probably lose weight; but in others harm may be done. As has been pointed out, there is no characteristic change in the basal metabolism of the obese, so that, as happened in a patient seen some years ago, hyperthyreoidism may be produced. Further, as has been shown, patients with frank myxœdema often do not lose much weight; in fact, in some cases the weight, after a preliminary fall, has been increased. However, where indicated, thyreoid substance has been used in conjunction with other measures. The initial dosage has been 0·03 gramme (half a grain) twice a day. If the patient tolerates this dose, an increase is made to 0·06 gramme (one grain) three times a day. Some patients, however, require much larger doses than this, and up to 1·0 gramme (15 grains) daily has been used in some of the myxœdematous cases. It must be remembered that the exogenous obesity is frequently associated with endogenous obesity, especially of thyreoid origin.

While the action of pituitary gland extracts given by mouth is uncertain, one of the patients to whom whole gland pituitary extract was administered did seem to benefit in that his retarded sexual development was accelerated; whether *post hoc* or *propter hoc* it was impossible to determine. The administration of whole gland pituitary extract to adults did not have any noticeable effect.

The combination of thyreoid substance and whole gland pituitary extract, as recommended by H. Gardiner-Hill,[13] was used in several cases and, as far as one could judge, was beneficial.

Treatment by extracts of the genital glands did not seem to have any effect.

Operative removal of large masses of adipose tissue recommended by some authorities was not adopted in the series of cases studied.

Results of Treatment.

Improvement was noted in many cases in which the thyreoid secretion was deficient, and good results were obtained in the exogenous cases when the patient was cooperative and realized that "the platter kills more than the sword".

Summary.

1. A short account of various aspects of obesity is given.

2. The paper is illustrated by reference to fifty cases of obesity.

3. The importance of diet and exercise in the treatment of obesity is stressed, and the indiscriminate use of thyreoid substance is condemned.

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(14) D. M. Dunlop and R. M. Murray Lyon: "A Study of 523 Cases of Obesity", *The Edinburgh Medical Journal*, New Series, Volume XXXVIII, Number 10, October, 1931, page 561.

APPENDIX.

Tables in which the Particulars of Cases are Summarized.

TABLE I.
Generalized Obesity (including Exogenous Obesity).

Case.	Age.	Sex.	Weight.	Height.	Blood Pressure.		Remarks.
					Systolic.	Diastolic.	
			St. lb.	Ft. in.			
1	20	F.	16 0	5 3			Obesity increased since birth of baby nine months ago.
2	28	F.	20 0	5 4	210	130	Following birth of twins weight increased in six years from 14 st. to 20 st. Complains of anginal symptoms. Mother (18 st.) suffered from *diabetes mellitus*.
3	61	F.	16 7	5 7			"Rheumatism" for years. Seven children.
4	57	F.	17 7				Osteoarthritis of the left knee.
5	44	F.	18 10 (27/6/29) 11 6½ (12/5/30)	5 5	140	90	Breathlessness and palpitation.
6	46	F.	11 3	5 4½	110	70	Swollen feet, stiffness of knees, backache.
7	60	F.	18 0		162	116	Breathlessness, cholecystitis, albuminuria.
8	52	F.	16 7	5 7½	120	80	Swelling of knees.
9	49	F.	13 5½ (5/12/29) 12 7½ (29/7/30)	5 4	150	100	Legs swollen. Last menstrual period in October, 1929.
10	50	F.	17 3 (27/4/33) 16 5 (21/9/33)	5 3			Ache in throat on exertion. Unable to sleep lying down. (Figures IA and IB.)
11	44	M.	18 8	5 6			Languor, increasing obesity, breathlessness.
12	22	F.	12 1½	5 1			Breathlessness.
13	36	F.	15 9 (19/3/31) 13 7 (31/3/31)	5 4	200 130	110 95	Breathlessness, swelling of feet, pain round heart.
14	48	F.	15 11	5 5	156	86	Pain in upper part of chest, worse on exertion. Weight increased by two stone in last twelve months.
15	41	F.	12 1½	5 5	120	90	Acute rheumatism.
16	36	M.	16 11 (14/9/32) 15 0 (21/8/33)	5 10½	94	70	Gout.
17	47	F.	17 11	5 0	150	100	Pain in left knee. Osteoarthritis. Hypothyreoid. Sister of Case 16.
18	17	F.	15 1½ (9/5/33) 13 5½ (8/8/33)	5 8½	130	86	Amenorrhœa.
19	43	M.	14 2½				Obesity following pneumonia.
20	49	F.	13 2½	5 4	124	76	Palpitation and increasing weight. Albuminuria. Oophorectomy seventeen years previously.
21	37	F.	15 12	5 5½	120	80	Pain and swelling in knee joints.
22	67	M.	15 11½	5 9½	110	78	Hotel-keeper. Gastric symptoms.
23	26	M.	12 10½	5 4½	114	80	"Nerves", vasomotor instability, over-indulgence in alcohol.
24	58	F.	16 6½	5 10	168	90	Arteriosclerosis.

TABLE II.
Obesity of Pituitary Type.

Case.	Age.	Sex.	Weight.	Height.	Blood Pressure.		Remarks.
					Systolic.	Diastolic.	
			St. lb.	Ft. in.			
1	48	F.	12 5½	4 9	120	80	
2	21	M.	15 5½ (24/1/30) 13 4½ (19/4/32)	5 4½	100	70	Breathlessness. Depressed bridge of nose, interstitial keratitis. Family history suggests congenital syphilis. No reaction to Wassermann test.
3	46	F.	12 9	5 2½	100	80	Died in coma. (?) Cerebral tumour.
4	24	F.	17 10	5 4½	120	76	Complained of obesity and irregular menstruation. (Figure IV.)
5	54	M.	22 0	6 0			Acromegaly, spondylitis, *sella turcica* wide.
6	28	F.	17 0 (22/12/32) 14 12 (14/9/33)	5 8	160	100	Pituitary fossa small. No evidence of erosion. (Figures IIIA and IIIB.)
7	15	F.	13 0	5 6½			Obesity commenced after removal of tonsils and adenoids two and a half years ago.
8	38	F.	18 8		130	90	Weighed 11 st. 12 lb. three years ago. Sella very small.
9	25	F.	15 7 (17/12/31) 16 7 (27/7/33)	5 0			Fits every three months. Weight increased from 8 st. 7 lb. five years ago.
10	14	M.	12 1½	5 4½	96	66	
11	25	M.	14 13½	5 9½	140	100	Weight has increased during last four years. Alcoholic gastritis.
12	15	M.	8 0 (23/5/32) 9 10 (10/8/33)	4 8			Delayed puberty; anterior and posterior clinoids close.
13	33	M.	17 10	5 0½	122	70	*Sella turcica* very small. Basal metabolic rate: −54%.
14	12	M.	9 9	4 11	120	80	Increase in weight in last six months. Pains in knees. Genitals normal.
15	15	F.	11 5½	5 5½	96	60	

TABLE III.
Obesity Associated with Myxœdema.

Case.	Age.	Sex.	Weight.	Height.	Blood Pressure.		Remarks.
					Systolic.	Diastolic.	
			St. lb.	Ft. in.			
1	50	M.	12 0 (28/10/26) 10 7 (13/1/27)				
2	57	F.	15 3 (15/5/33) 14 8 (21/9/33)		150	94	Basal metabolic rate: −43%. Distribution of obesity suggests pituitary element.
3	62	F.	14 6½	5 3½	120	80	Has been under treatment for about two years.
4	46	F.	16 7				
5	54	F.	11 4		188	110	Weight had increased from 8 st. 8 lb. before onset of myxœdema five years ago. Decreased with treatment.
6	30	F.	11 7		130	110	Weight increased while under treatment.

TABLE IV.

Obesity Apparently Following Oophorectomy.

Case.	Age.	Sex.	Weight.	Height.	Blood Pressure.		Remarks.
					Systolic.	Diastolic.	
1	35	F.	St. lb.	Ft. in.			Double oophorectomy four years previously. Increase of three stone in weight. Eight children, three miscarriages.
2	32	F.	10 9	5 5	150	95	Section five years ago. Increase in weight from 9 st. 6 lb. to 16 st. 7 lb. Adiposity in region of hips.
3	37	F.	10 11				Double oophorectomy five years previously. Increase of weight from 7 st. to 10 st. 11 lb. Cholecystitis.

TABLE V.

Obesity Associated with Endocrine Disturbance of Ill-Defined Type.

Case.	Age.	Sex.	Weight.	Height.	Blood Pressure.		Remarks.
					Systolic.	Diastolic.	
1	59	F.	St. lb. 20 0	Ft. in. 5 2	190	100	Palpitation, arteriosclerosis. (?) Thyroid, gonad and pituitary obesity.
2	32	F.	14 6	5 6	140	100	Amenorrhœa for ten months, followed by flooding. Trochanteric and abdominal obesity. Pubic hair of male type. (?) Suprarenal or pituitary involvement. (Dr. A. R. H. Duggan's patient.)

Reports of Cases.

STRYCHNINE POISONING IN A CHILD.

By CHAS. PERRY,

Honorary Consultant to First Victorian Training School of Mothercraft and Hospital for Babies.

THE following case is, I think, of sufficient interest to justify reporting.

On Saturday, February 17, 1934, I was asked to go to a good country home about ninety miles from Melbourne to see a baby, aged three months.

The baby had previously been in good health. It was being breast fed and was having complementary cow's milk.

The recorded history was that quite suddenly, at 4 a.m., the baby had been seized with convulsions. The convulsive seizures were recurring repeatedly at intervals of half an hour for the first three hours. The intervals then lengthened, and when I arrived at midnight, there had been no attack for four hours. There was no vomiting or any intestinal disturbance.

At 1 a.m. the child had an attack which, on observation, I was satisfied was not a convulsion in the generally accepted form. It was more in the nature of a general muscular spasm resembling tetany, but not typical. The tests for tetany gave negative results. I was then troubled to explain the condition in a satisfactory manner, but decided it was due to some form of poison, probably of a chemical character.

I then made inquiries as to the possibility of milk infection, and found that the dairy cows were grazing in a paddock where a lot of poisoned rabbits had been burnt. The milk was submitted to Mr. A. H. Koch, a qualified analyst and public analyst to various municipalities. He reported the presence of traces of strychnine in both the cream and milk. The child was, of course, taken off the complementary feeds immediately my suspicions were aroused.

On Sunday, when I left, the child had not had any twitching for six hours. For the next twenty-four hours occasional twitchings were reported, but had entirely ceased by Tuesday.

On Wednesday I again saw the child, and it was perfectly well, though somewhat pale. Unfortunately the shock and strain caused a complete cessation of the mother's milk secretion.

I then worked out a modified milk mixture based on Sir Truby King's method.

The progress was in every way normal until Saturday, March 3, when I was again called to see the child, as there had been a return of the seizures, but of a much milder nature.

On arrival I found the condition exactly similar, though the spasms were not nearly so severe or so frequent. The tetany tests again gave negative results.

The cows had, of course, been transferred to another paddock and I was then unable to associate the condition with a similar cause. However, the milk and cream were again submitted to Mr. Koch, and he reported a trace of strychnine in the cream, but not in the milk. The cream submitted was from Friday's milking, but the milk was of a later date.

The child was then brought to town and placed in hospital and put on a modified milk mixture suitable for its age and weight. It has gone straight ahead without any return of the twitching.

My reason for reporting the case is that perhaps some of my *confrères* may have met with cases occurring under similar conditions and of a similar nature, particularly if occurring at this time of the year, when much strychnine is being used for rabbit poisoning.

In Victoria it is compulsory to poison rabbits during the months of January and February.

Reviews.

A TEXT BOOK ON HISTOLOGY.

ALTHOUGH Ramón y Cajal is probably the most famous of living histologists, he has been somewhat handicapped in publishing his views in the scientific world on account of the fact that he has not been able to write in his native language. Most of his work, and that of the brilliant school of which he is director, has appeared in the French. For that reason Cajal's work has not been so accessible to the English reader. It is with particular pleasure,

therefore, that we see a translation of Cajal's text book, which more or less represents a compendium of the investigations of the illustrious author and his disciples, and which, moreover, gives us an opportunity of seeing how the great master presents his subject to his classes.[1] The translator hopes that this work "will serve as a guide to Spanish histology and inspire the reader to investigate more deeply Spanish medical literature, with the assurance that a veritable mine of pleasure awaits him". The present work is a translation of the elementary student's text book, as used in all Spanish-speaking universities, in which the system of instruction differs somewhat from that obtaining in English-speaking countries. The translation closely follows the original text, and the nomenclature, phraseology and syntax, often appear a little unusual. The translator has followed this procedure with the set purpose of making English readers familiar with many of the vivid teaching locutions so characteristic of Cajal's lecture room.

The first seven chapters are devoted to cytology, and represent a very thorough presentation of the most important facts of this difficult branch of histology. Of the remaining sections, those which deal with muscular and nervous tissues call for special comment. These subjects receive a fuller treatment than is usually attempted in an elementary text book, that of the nervous tissue alone occupying two hundred pages. This is to be expected, as it is just in this branch of histology that Cajal is so famous. Those familiar with his eminence will welcome this compendium compiled by Cajal himself.

All other sections of the work are adequately treated and are comparable with similar sections in other first class text books. A large chapter and appendix are devoted to histological methods. This is especially valuable as the main procedures for the preparation of material, as used in the Cajal laboratories, are given in adequate detail. The whole work is profusely illustrated with large, clear and informative figures, and a great number are either original or are products of Cajal's disciples and set a very high standard as text book illustrations.

INHALATIONAL THERAPY.

IN "Modern Inhalation Therapy for the General Practitioner", the author, C. H. Auty, M.R.C.S., offers his personal experiences.[2] He is an ardent enthusiast for the administration of certain drugs (as iodine, creosote, strychnine, brandy *et cetera*) in the form of a nebula produced by oxygen or air.

The author is scientifically honest and makes no sweeping claims. He merely records his personal experiences in the inhalational treatment of conditions so diverse as anæmia, bronchitis, gout, heart disease, pneumonia, prostatic enlargement, tuberculosis and varicose veins. Some of these groups embrace only two or three cases. His criteria of benefit are based on personal clinical impressions; he makes no attempt at biochemical or statistical analysis or scientific "controls". His faith in the value of drugs, and in the superiority in certain cases of their administration by inhalation rather than orally or hypodermically, is a matter of personal conviction only. Certainly, he makes no allowance for the psychological effect of inhalational therapy and for the influence of the oxygen by which the drugs are vaporized, factors which in some of his cases are no less to be considered than the action of the drugs themselves.

The little volume may perhaps do good by reminding us that inhalational therapy has been unduly neglected. Its claims have been ignored, or, in some instances, uncritically accepted. Even the traditional faith of clinicians in such pulmonary antiseptics as creosote should be put to critical proof. The proof, however, must rest on data more scientifically "controlled" than those of Dr. Auty, whose zeal would seem to have disarmed his critical faculty.

In his preface, Dr. Auty claims to describe treatment "possibly a little in advance of many of the accepted principles of the present day". In view of this claim, it is surprising to find the whole question of modern oxygen therapy dismissed in a few slighting lines, whilst carbon dioxide therapy is accorded no mention at all.

PSYCHIATRY FOR THE PUBLIC.

"THE LAST OF THE TABOOS", by Dr. Isabel Emslie Hutton, is written in crusading spirit to break down the still prevailing public prejudice in regard to mental maladies.[1] The style is frankly popular, and the book is intended to be read by the general public, and in particular by magistrates, social workers, politicians, clergy, journalists, and medical men who might be expected to take a lead in reforming public opinion.

The author describes, in simple language, the various forms of mental maladies, and proceeds in successive chapters to explain present day methods of care and treatment.

Special consideration is given to the difficult subject of mental deficiency, and the author discusses the pros and cons of sterilization, which she advocates as a partial solution for the problem.

In the chapter on legal aspects of mental disorders, Dr. Hutton gives a valuable *résumé* of the law of England relating to mental hospitals and to the certification and treatment of the mentally disordered. In conclusion, the author makes suggestions for the amelioration of the lot of the person suffering from a mental malady.

Whilst adding nothing new to our knowledge, this book may be recommended confidently to the public as a reliable exposition of the present day status of psychiatry.

THE PHYSIOLOGY OF THE EYE.

A MOST interesting volume by J. Grandson Byrne has appeared. It is a report of hundreds of experiments that he performed, and of his deductions from them in attempting to explain many problems in ocular and general physiology.[2] The experimental work has been carried out in laboratories in England, Spain, Germany, and France. "With the exception of a chapter on the functions of the cervical sympathetic as manifested in its action currents, which was done in collaboration with Professor William Einthoven at the University of Leiden, the experimental work is solely the product of the author's own thought and labour."

There are four divisions in the book. The first has been published in the *American Journal of Physiology*, 1921 to 1929, and deals with the parodoxical pupillary phenomena following lesions of the afferent paths. Throughout the book the various topics are introduced by a brief survey of the literature. This is followed by a report of the author's experiments, and a discussion of their significance. A summary of the findings and a bibliography conclude each chapter. In the first chapter, numerous experiments

[1] "Histology", by S. Ramón-Cajal, M.D., F.R.S., LL.D., revised by J. F. Tello-Munoz, M.D., authorized translation from the tenth Spanish edition by M. Fernán-Núnez, M.D.; 1933. London: Baillière, Tindall and Cox. Royal 8vo., pp. 752, with 535 illustrations. Price: 40s. net.

[2] "Modern Inhalation Therapy for the General Practitioner", by C. H. Auty, M.R.C.S., L.R.C.P.; 1933. London: William Heinemann (Medical Books) Limited. Crown 8vo., pp. 78, with illustrations. Price: 3s. 6d. net.

[1] "The Last of the Taboos: Mental Disorders in Modern Life", by I. E. Hutton, M.D.; 1934. London: William Heinemann (Medical Books) Limited. Crown 8vo., pp. 217. Price: 6s. net.

[2] "Studies on the Physiology of the Eye: Still Reaction, Sleep, Dreams, Hibernation, Repression, Hypnosis, Narcosis, Coma, and Allied Conditions", by J. G. Byrne; 1933. London: H. K. Lewis and Company, Limited. Royal 8vo., pp. 425, with 48 illustrations. Price: 40s. net.

are reported in which lesions of various afferent pathways were made, and the resulting pupillary changes were observed. "The functional relationship between the periphery and the pupil dilator mechanism through the sensory nerves is such that tissue injury of all sorts or degrees may cause inequality of the pupil. Care should therefore be taken when interpreting clinically the significance of unequal pupils, to give due consideration to possible injuries of the afferent paths in the peripheral nerves." Therefore the inequality of pupils associated with injuries of or near the brachial plexus need not necessarily be due to a lesion of the cervical sympathetic nerve. The sensory nerves of the head, neck, upper extremity *et cetera* are preponderantly related to the homolateral pupil, whereas those of the hindquarter are preponderantly related to the contralateral pupil.

Lesions of one-sided organs which have an afferent supply entering the spinal cord above the tenth thoracic segment are mainly associated with paradoxical pupil dilatation in the homolateral eye. Organs with an afferent supply reaching the cord caudad to this level are associated with the contralateral eye. One conclusion at the end of Chapter VIII is that the proprioceptive paths from the external ocular muscles run exclusively in the ophthalmic division of the fifth cranial nerve.

Part II deals with preliminary palpebral widening, paradoxical palpebral and lens phenomena and inherent tonus phenomena. The findings are largely parallel with those of Part I.

In Part III there is a description of various stimulation experiments made to determine the extent to which reciprocal innervation obtains between the affectivo-sympathetic mechanisms which mediate proptosis and palpebral, pupillary and lens widening, and the critical proprioceptive-parasympathetic mechanisms which mediate enophthalmos and palpebral, pupillary and lens narrowing. The significance of the results of the experiments is discussed.

The inherent constrictor tonus of the oculomotor nerve and the mechanism of ether narcosis, coma, sleep, the still reaction and allied conditions are discussed in Part IV. Little is understood regarding the constriction of the pupils during sleep, coma and the allied conditions. An endeavour is made by the author to show that this constriction, as well as the conditions in which it occurs including "catalepsy, narcolepsy, hysteria, *et cetera*, are release phenomena, like decerebrate rigidity, with which it is closely related functionally and developmentally". Most interesting descriptions of the death-feigning or still reaction of hibernation, and of sleep follow. The author considers that the still reaction can be utilized as a means of controlling restless animals in the laboratory, of facilitating the physical examination of restless children and of putting wakeful infants to sleep.

Three stages in the development of occular effector mechanisms are discussed. They are the primitive type in which activation is effected by chemical means, the intermediate or chemico-neural type in which neural impulses are initiated in the related centre, and the higher level at which the effector mechanisms are of the reflex type and in which both afferent and efferent impulses are active.

This book should be a valuable addition to the library of any worker interested in the problems of ocular physiology. The illustrations and diagrams are well chosen and clear. The mode of production is good.

EMBRYOLOGY.

MANY teachers in British universities will welcome the fifth edition of Sir Arthur Keith's "Embryology and Morphology".[1] It is ten years since the last edition was published and quite considerable advances have been made in embryology since that time, particularly in the earlier stages of human development and in experimental embryology. Among text books of embryology written for medical students, Keith's manual is unique in its method of approach as the author is convinced that "embryology becomes a profitable subject of study only when its facts are interpreted in the light of comparative anatomy". He points out that information concerning the history of the human body is obtained from two sources, namely, the embryological and the morphological or evolutionary, and he has therefore drawn his facts for his book from both historical sources. It has long been recognized that for a complete understanding of human anatomy, facts from both sources are essential; but few embryologists have been qualified to bring them into one text book such as Keith has done. In the first edition of this work, clinical utility was the criterion employed in the selection of facts, but the author's experience led him to depart from this narrow utilitarian ideal and to include in subsequent editions all those facts which helped the medical student to understand better the human body. This was a wise policy, as the book in its present form must be regarded as one of the most valuable of the elementary British text books of embryology. Obviously such a wealth of subject matter can be dealt with only in an elementary way, but the book serves as a clear and easily followed introduction to all topics which are likely to present themselves to students of anatomy.

Copious references are provided which enable the student to find more detailed accounts, and further, many of these are discussed and annotated by the author. Probably nowhere is the correlation of embryology and comparative anatomy more illuminating than in the account of the central nervous system; but it is difficult to discriminate between the various sections. Others which are particularly well done are those on the skull, face, pharynx, neck, heart and the urino-genital system. A new chapter has been added dealing with the recent advances in experimental embryology, and numerous alterations in the text are noticed throughout. The work is fully illustrated with simple and yet very effective figures, numerous additions and replacements being made for this edition. The number of illustrations has been increased from 490 to 535, while eighteen of the old ones have been redrawn or replaced. Altogether, the book has been completely brought up to date and should continue to enjoy its full measure of popularity.

[1] "Human Embryology and Morphology", by Sir Arthur Keith, M.D., F.R.S., LL.D., D.Sc., F.R.C.S.; Fifth Edition; 1933. London: Edward Arnold and Company. Royal 8vo., pp. 566, with illustrations. Price: 32s. 6d. net.

Notes on Books, Current Journals and New Appliances.

THREE SHORT HISTORY BOOKS.

THREE volumes of the "Short History Series" have been received. The volume on ophthalmology is from the pen of Dr. Arnold Sorsby.[1] The volume on epidemic infectious diseases is by Dr. E. W. Goodall,[2] and that on physiology is by Dr. K. J. Franklin.[3] The publishers are to be congratulated on producing these short volumes. Medical practitioners who have neglected the study of medical history, who are busy, and who wish to know something about the subject, will find these volumes of use. They are of a convenient size and can be carried in the pocket.

[1] "A Short History of Ophthalmology", by A. Sorsby, M.D., F.R.C.S., with foreword by R. R. James, F.R.C.S.; 1933. London: John Bale, Sons and Danielsson, Limited. Crown 8vo., pp. 109, with illustrations. Price: 3s. 6d. net.

[2] "A Short History of the Epidemic Infectious Diseases", by E. W. Goodall, O.B.E., M.D.; 1934. London: John Bale, Sons and Danielsson, Limited. Crown 8vo., pp. 113. Price: 3s. 6d. net.

[3] "A Short History of Physiology", by K. J. Franklin; 1933. London: John Bale, Sons and Danielsson, Limited. Crown 8vo., pp. 122. Price: 3s. 6d. net.

The Medical Journal of Australia

SATURDAY, JUNE 23, 1934.

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References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

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PREVENTIVE SURGERY.

SURGERY, strictly speaking, is the art or practice of treating injuries, deformities and other disorders by manual operation or instrumental appliances. It is, however, more than carpentry of the human body. To give adequate treatment to a maimed or disordered body a surgeon must understand the physiological functions of the healthy body; he must know the variations, the vagaries, that come within the range of the normal; he must be able to recognize incipient pathological change; he must understand the origin and development, as well as the significance, of a pathological lesion. A mere carpenter would have his diagnosis made for him by the practitioner who called him to see the patient, and would operate without further ado—like a good tradesman, he would be given the specifications of his job and would demolish, alter or try to build up the structure affected, as he was called upon to do by the person drawing up the specifications. The surgeon is rightly jealous of the range of his activities. Although much of his pre-operative and post-operative treatment is dependent on the use of apparatus, he approaches his problem, or he should approach it, with the mind of a physician; and he adopts measures, ofttimes simple, that are likely to be regarded as being within the physician's sphere of activity. During the last decade or two surgeons specialising in the treatment of certain conditions have extended the range of their knowledge and activities. For example, the neuro-surgeon combines delicate dexterity with the neurological investigation of the physician; the thoracic surgeon enters the uncertain world of physical signs and takes adventurous steps in their interpretation with as much success as the physician; the urological surgeon devotes as much time and study to the renal function as the physician is wont to do. But the surgeon has to go still further afield. Dr. F. P. Sandes stated recently at the Hobart Congress (and the words carried conviction) that if the surgeon wished to be expert in cancer treatment, he should not only be able to assess the value of the opinion of over-enthusiastic operators, radiotherapists, pathologists and research workers, but should learn something about the nature of electro-magnetic waves and their biological effects, and have a working knowledge of laboratory investigations applicable to cancer pathology. Surgery has in fact become a complex science in which manual dexterity and the use of appliances are merely a part, and sometimes a lesser part.

A great deal has been written from time to time in this and other journals on the value of preventive as opposed to curative medicine. Not enough has been written about preventive surgery. Many surgical operations are frankly curative—the removal of a gangrenous appendix from a desperately ill patient, the amputation of an injured limb already affected by gas gangrene, the removal of a Fallopian tube in a case of ruptured ectopic gestation. Even here, however, there is an element of prevention—if the appendix is not removed, general peritonitis will follow; if the limb be not amputated, the gas gangrene will spread to the rest of the body; if the Fallopian tube be not removed, further hæmorrhage may cause death of the patient. In certain operations prevention appears to be more obvious. An inguinal hernia is operated on so that strangulation of the gut will not occur. The round ligaments of the uterus are shortened and the *levator ani* muscles in the pelvic floor are repaired so that uterine prolapse will not take place. Tonsils are removed so that systemic infection will not

result from their infected crypts. And so on. The surgeon may thus lay the unction to his soul that in most of his operative work he is preventing disaster as well as curing an abnormal condition.

Since the surgeon's work is largely that of the physician, he should aim at carrying his efforts at prevention further than he does with his operative measures. A surgical operation is an ordeal to most people; and though the mutilation that possibly results may be a signal of surgical prowess, it may be a constant companion of the unhappy patient. Sometimes both the mutilation and the ordeal may be avoided. It is commonly said that unnecessary surgery is bad surgery; the same is, of course, true of avoidable surgery. If by carefully planned non-operative treatment the patient can be restored to health, operation should be avoided, even though operation would achieve the same result. This is realized by most men doing surgical work, but not by all; chief among the purblind is the tiro seeking his reputation at the scalpel's edge. The best corrective for such an aspiration is a tirocinium spent first in general practice and then in subservience to a senior surgeon of experience. In a country where any medical practitioner can call himself surgeon and none has the right to gainsay him, it is still necessary to declare, even though it be a platitude, that the aim of preventive surgery should be the avoidance of surgery.

Current Comment.

DISPOSAL OF NIGHT-SOIL.

WHERE there is no sewerage system the proper disposal of human excreta is difficult. There are many things to be considered apart from the mere removal of noisome material from the regions of dwellings, though this is all that interests the majority of citizens, and certainly it is urgent enough in many places. Unfortunately, disposal of night-soil is generally regarded as a disgusting business, almost unfit to be discussed. Persons engaged in it are placed so low in the social scale that they are not mentioned in polite circles. Their trade is plied under cover of darkness while the citizen sleeps on. This lack of interest is only one of the reasons why the methods of night-soil disposal in many places have not improved commensurately with improvements in other measures of hygiene and sanitation. There are very few towns in Australia where every householder's night-soil is removed more frequently than once a week: quantities of fæces that have accumulated for a week are perhaps no more dangerous than fresh fæces; but they are distinctly more unpleasant. Lack of funds may be given as the reason for this dilatoriness; but the work should be done with sufficient economy to make funds available. This is possible if regard is had for the economic uses of night-soil. For centuries the Chinese have utilized human fæces and urine for manuring their gardens; but their methods are unhygienic and tend to encourage the dissemination of parasitic and other diseases. In parts of India farmers collect refuse and night-soil from towns, and after allowing the mixture to rot, spread it on their land. Human waste is recognized by these people as the best of all fertilizers. It is by such means as the preparation and sale of fertilizers that the disposal of night-soil can be economically effected; in fact, it has been found possible to conduct the business at a profit rather than a heavy loss to the civic authorities.

F. K. Jackson and Y. D. Wad, of the Institute of Plant Industry, Indore, central India, have recently described a method, known as the Indore method, which is simple, cheap and efficient, and can be a source of municipal income.[1] They quote Leather (1895) as follows:

> There is, perhaps, no more important subject in relation to agriculture than the proper disposal of night-soil and other town refuse, for it may be said, without exaggeration, that nearly one-half the plant food extracted by food crops from soil is contained in the materials which are included under these two heads. It follows, therefore, that on their proper disposal and return to the soil depends the addition of a large proportion of the food necessary for the crops.

The Indore method depends on the scientific composting of refuse and night-soil. A trench is made, 4·5 metres (fifteen feet) wide, 0·6 metre (two feet) deep, and as long as required. It is floored with road metal or brickbats and rubble, and its banks are supported by logs or stones; it must have a gentle slope towards a natural drainage channel. A layer of refuse, 7·5 or 8·0 centimetres in depth, is spread across the bottom of the trench; a cartload of night-soil is tipped in, and a load of refuse immediately follows; the two are then raked across the trench to form a layer over the layer of refuse below. About four layers a day of this night-soil and refuse mixture are added, the top of the mass reaching the level of the top of the trench in two days. At the end of each day the top is covered with a layer of refuse alone. The top is made flat and the sides vertical. Two days later the mass is turned with a four-pronged digging fork; this is repeated at the end of a further eight days. Eight to fifteen days later, depending on the rapidity of decomposition, the whole mass is turned out of the trench and stored in the open ready for sale. In dry weather it may be necessary to add a little water occasionally to the mass in the trench. Rubbish of all kinds is included in the refuse; no proportioning of refuse to night-soil is necessary,

[1] *The Indian Medical Gazette*, February, 1934.

all being deposited in the trench as it arrives at the disposal ground.

Jackson and Wad point out that night-soil tends to liquefy when it is allowed to accumulate, and gives off foul volatile products. If an attempt is made to mix night-soil intimately with refuse "it fills the interspaces and forms a dense, viscous, anaerobic mass, thus preventing oxidation of the noxious products which continue to be formed at an intensified rate". But if, as in the Indore process, the night-soil is spread rapidly with the refuse in thin layers, it "forms a coating on the refuse with wide interspaces for aeration"; furthermore, the partial desiccation that follows clots the colloids and slows their decomposition. Thus the formation of obnoxious substances is slowed, and as they are formed the adequate aeration provides for their immediate oxidation. These are the reasons why there is no nuisance with the Indore method.

The temperature in the mass is from 45° C. to higher than 50° C. This destroys pathogenic bacteria and prevents fly breeding. At each turning any larvæ that have hatched out at the edges are thrown into the middle and rapidly killed. The periods between the turnings are short enough to prevent larvæ from maturing and reduce their chances of crawling into crevices at the sides of the trench to pupate.

Difficulties that will at once suggest themselves to the expert in sanitation are discussed in the paper, which should be read by all health authorities. Among the advantages of the Indore method may be mentioned: low capital outlay and running expenses; economy in the area of the disposal ground; permanency of the trenches, which are constructed simply without special engineering skill; prevention of offensive odours and fly breeding; the provision for rapid and efficient disposal of refuse without the necessity for elaborate structures; applicability to large or small communities. Other advantages are readily suggested. It may be added that Jackson and Wad believe that the method would be suitable in "a wide range of climates and agricultural conditions".

The Indore process might be tried in Australia and the Australian territories. No doubt there would be considerable prejudice to be overcome before the method could be widely employed in this country. People will use horse and cattle manure and various rotting and filthy materials for fertilizing their soil; but they will turn aside in disgust at the suggestion that any product of human excreta should be so employed.

THE ELECTROCARDIOGRAPHIC DIAGNOSIS OF CORONARY OCCLUSION.

THE electrocardiograph as a special technical aid in the diagnosis of diseases of the heart was first restricted to the elucidation of the irregularities. Latterly, however, its true importance has proved to be concerned with the electrical changes observed in nutritional diseases of the heart. Acute infarction of the heart is now a well known syndrome, and where the signs and symptoms are clear cut and dramatic, as they often are, the clinical picture is well nigh unmistakable. In these cases no instrumental aid is required, but not all instances of this serious accident declare themselves so plainly. In the doubtful and difficult cases, of which there are many, the study of the electrocardiographic record is of great assistance. Still more difficult is it to estimate the condition of the coronary blood supply, and of the cardiac muscle in ambulatory patients who complain of symptoms that are mild, though suspicious. Here the physician's diagnostic acumen is tested, for such symptoms are not infrequently referred to the digestive tract. This was found to be so in many of the patients studied by J. A. Goldbloom in a contribution towards the clinical evaluation of the fourth lead in the electrocardiogram.[1] Practically all the patients were ambulatory, and half of them presented definite clinical signs of coronary artery disease. The object of the inquiry was to discover the value of this recently introduced fourth lead. He wished to find if this lead gave reliable information obtainable by none of the standard leads, and if so, in what proportion of cases.

A. J. H. Stobo has recently described in this journal results obtained by preliminary trials of this method, which was found to give very promising diagnostic assistance. Goldbloom's figures clearly indicate that a greater degree of accuracy can be attained by the use of this additional lead. He is careful to point out that the lead must always be used in a standard fashion, but finds that a simple technique is best; he rubs the skin of the back and the chest with warm saline solution, and bandages the electrodes firmly in place, but uses no special appliance. He finds that the clinical diagnosis (of which he seems to be reasonably confident, though he gives no details) was sustained by the ordinary three-lead record in 70%. The fourth lead was found to correspond very closely with the others, though an agreement in all leads was observed only in 30%. However, in 7·5% he found definite evidence in the fourth lead alone, the other leads giving normal graphs. Also in 30% of a small series of cases of acute cardiac infarction the fourth lead alone testified to the change that had occurred. This confirms what others have written. It is known that electrocardiography, like every other diagnostic method, cannot be expected to be 100% correct, but the more thorough the investigation, the higher the percentage of accurate positive findings. It is worth while, therefore, to include this antero-posterior lead in routine electrocardiographic examination when the myocardium is on trial. Another practical reason may be added. A negative report of any kind by any special method is apt to be misinterpreted by the patient, and sometimes even by the doctor, in spite of the disclaimers of the expert, and any additional degree of accuracy attainable in any scientific method of diagnosis is all the more to be welcomed.

[1] *The American Journal of the Medical Sciences*, April, 1934.

Abstracts from Current Medical Literature.

MEDICINE.

Fat and the Diabetic.

E. P. JOSLIN (*The New England Journal of Medicine*, September 14, 1933) discusses the rôle of fat in relation to diabetes. The Naunyn era, 1898-1914, was the era of uncontrolled fat diet, the carbohydrates alone being restricted; it was associated with a death rate from coma of 60%, while the average diabetic lived 4·8 years. In the Allen era, 1914-1922, there was restriction of the total calories, but the fat content of the diet was still high, the patient lived for about six years, and the deaths from coma were about 40%. Nowadays, the era of Banting and Best, the expectation of life approaches normality and the death rate from coma is less than 5%. Heredity has replaced obesity as the most significant factor in the ætiology, but the families of patients should be warned against becoming fat. The fat individual has a lowered carbohydrate tolerance and fat foods lower the efficiency of the pancreas just as effectively as carbohydrates. The effect of exogenous and endogenous fats is similar. The person who loses weight rapidly is living on a high fat diet of his own fat. Rapid development of obesity is more dangerous than gradual development, and occasions leading to rapid increase in weight, such as the beginning and end of the sexual cycle, change of occupation, and the enforced idleness due to fractures, must be foreseen. Inanition leads to a greater deposition of fat in the liver, derived from body fat, than does fat feeding. Deprived of its pancreas, a dog dies in one or two weeks. Though life may be prolonged for several months by giving insulin, ultimately the liver function fails, glycosuria ceases, and towards the end hypoglycæmia has been noted which persists even if the insulin is then withdrawn. This sequence of events is accelerated if fat be added to the diet. If raw pancreas be given in the diet in addition to the administration of insulin, the animal may be kept alive indefinitely. Lecithin may be used to replace the raw pancreas, and even when the animal is almost moribund after insulin only lecithin will bring it back to life and it again begins to excrete sugar. With the aid of lecithin fatty acids become desaturated and available for use in the body. The action of lecithin depends on choline, one of its constituents. Choline is toxic, but its non-toxic oxidation product, betaine, acts as well. The quantity of sugar in the urine resulting from the lecithin is greater than could be derived from glycogen and from the protein metabolized. The question arises as to whether this sugar comes from the lecithin, in other words, from fat. Fat in the blood exists in three forms: fatty acids, free and combined, lecithin and cholesterol. Cholesterol is universal in its occurrence and is a vehicle for the transfer of unsaturated fatty acids in the blood stream. In the diabetics of 1916 it was far above normal. Nowadays it may be well within limits owing to the introduction of insulin. Fat is a far more insidious factor in the blood of a diabetic than sugar. Cholesterol does not stand in relation to sugar in the blood. The normal range of cholesterol is from 125 to 230 milligrammes per 100 cubic centimetres of blood: a blood cholesterol of 300 milligrammes should be viewed with alarm as presaging complications.

Chronic Pulmonary Infection in School Children.

BANKS AND WEIR (*Tubercle*, June, 1933), as a result of the study of one hundred children between the ages of five and fourteen years admitted to a sanatorium, decided that while tuberculous disease of the lung is very rare, non-tuberculous pulmonary infection is very common in children, but that the latter disease is commonly diagnosed wrongly as tuberculosis of the lungs. Fifty-eight children reacted to a cutaneous tuberculin test, and forty-two failed to react. Sixty-six cases were finally diagnosed as chronic pulmonary catarrh. In twenty-three the diagnosis of chronic bronchitis was made. Ten patients were suffering from malnutrition without any respiratory lesion. One patient had pleurisy with effusion, this being the only case in the series in which evidence of recent tuberculous disease was obtained. As a practical application of their findings they offer the following points: (i) That children between the ages of three and twelve should not be considered as suffering from pulmonary tuberculosis in the absence of positive findings, such as tubercle bacilli in the sputum, or of X ray evidence of tuberculous infiltration. (ii) That the non-tuberculous catarrhal and fibrotic process, generally termed "chronic pulmonary catarrh", should be recognized as a sequel of measles, pertussis and bronchopneumonia. (iii) That the open-air school, and not the sanatorium, is the appropriate treatment for this condition. (iv) That more attention should be paid to prophylaxis, especially by treating children with pneumonia in hospital and by insuring abundance of vitamins during infancy and convalescence.

Radiography of the Liver.

W. M. YATES AND L. S. OTELL (*Annals of Clinical Medicine*, September, 1933) describe the technique of Röntgenography of the liver and spleen after intravenous injection of thorium dioxide sol or "Thorotrast". Eighty patients were investigated. Twenty-five cubic centimetres of "Thorotrast" were injected intravenously in average adults on each of three successive days. Films were taken at any time after four hours following the last injection. In good films the liver should cast a homogeneous shadow of about the same density as the spine; the spleen a less dense shadow. The right lobe of the liver was usually much more obvious than the left lobe. Alterations in the liver and spleen shadows enabled the diagnosis to be made or confirmed in cirrhosis of the liver, metastatic carcinoma of the liver, syphilis of the liver, and primary tumour of the liver, and in a variety of other disorders of the liver and spleen. The method was also useful in excluding subphrenic abscess and rupture of the spleen. No serious reactions were observed; transient lumbar pains, chills, moderate fever and a feeling of tension in the limbs were noted on a few occasions; however, in view of the possible dangers, the authors suggest that at present the method be used only for patients who are the subjects of a rapidly fatal disease.

Polycythæmia.

E. H. FALCONER (*Annals of Internal Medicine*, August, 1933) discusses the treatment of polycythæmia, with special reference to the reticulocyte response after venesection, phenylhydrazine and radiation. The normal reticulocyte percentage was between 0·4 and 1·5. Four patients with polycythæmia were treated by irradiation of the spleen and long bones; the reticulocyte response was observed, and at a later date a similar observation was made following treatment with acetyl phenylhydrazine or phenylhydrazine hydrochloride, 0·1 gramme daily for fourteen days, up to 4·0 grammes in sixteen days. In one patient venesection was performed ten times in five months; up to 600 cubic centimetres of blood were withdrawn on each occasion. Each method of treatment was followed by improvement in the symptoms at times; but phenylhydrazine caused weakness, mental confusion, giddiness and numbness of hands and feet in two patients, and the reticulocytes rose to 8% in one patient, indicating a stimulating effect on the bone marrow. Neither venesection nor irradiation caused an increase in the reticulocytes, but the action of irradiation was slow. The author concluded that venesection was a useful adjunct to phenylhydrazine treatment, enabling small doses to be used with less danger to the patient.

The Cause of Death in High Intestinal Obstruction.

J. E. SWEET (*New York State Journal of Medicine*, October 15, 1933) believes that a toxic substance, perhaps histamine-like, not normally found in the intestinal tract, arises under conditions of obstruction which injures the adrenals. The intestine of an animal after bilateral adrenalectomy without obstruction seems to contain the same toxin. In obstruction a vicious circle is set up; the

greater the injury to the adrenal, the greater the production of toxin, so that unless the removal of the primary cause be carried out before the adrenals are damaged too extensively the vicious circle may continue, with the result that the operation is futile. He points to the similarity between acute pancreatitis and high intestinal obstruction, between which few would venture a differential diagnosis. Both are strikingly unlike the clinical picture of the shock following excessive loss of blood. There is not in every case of obstruction any great loss of salt and water from the body by vomiting. Conversely, Asiatic cholera, for instance, in which there is a much more pronounced loss of body fluid, is not accompanied by any of the symptoms characteristic of the picture under discussion. The condition is comparable with anaphylaxis, in which a very small dose of foreign protein sets up a fatal process in a sensitized animal. If the contents of an obstructed loop of bowel be extracted with hot water, precipitated in an excess of alcohol, redissolved in hot water and again precipitated with alcohol, a highly toxic product is obtained which, injected into a normal animal, may reproduce the complete picture of obstruction. But though two animals as nearly alike as possible be treated with the same dose of this poison, one may react but slightly, while the other may be dead in six hours. From the intestine of an animal killed in this way a similar toxic product can be obtained by treating the contents in the same way as those of the original animal, though there has been no mechanical obstruction. There is no doubt that loss of water from the body aggravates the condition and that addition of water and sodium chloride is a life-extending procedure. It is, however, questionable as to how many of the chemical changes in the blood are primary and how many are secondary.

Acute Extrarenal Azotæmia.

M. RACHMILEWITZ (*The Lancet*, January 13, 1933) reports five cases of uræmia of extrarenal origin. All were of sudden onset following excessive loss of fluids and salts from the gastro-intestinal tract in patients suffering from cholelithiasis (vomiting and diarrhœa), duodenal ulcer (persistent vomiting), acute gastro-enteritis, vomiting of cerebral origin, and uncontrollable diarrhœa. In all the usual signs of kidney involvement, such as changes in the *fundus oculi*, increase of blood pressure and anæmia were absent. The blood pressure was low at the height of the condition and increased gradually with improvement. After administration of chlorides and glucose the patients recovered rapidly, with the exception of the patient with uncontrollable diarrhœa, who died. In one the blood urea dropped from 337 milligrammes *per centum* to 10 milligrammes within a few days. At the height of the azotæmia oliguria was observed. After the administration of chlorides and glucose with fluids there was a diuresis. Parallel with the oliguria a low excretion of chlorides in the urine was observed, while the urea and uric acid output was normal or rather increased. The significance of the chloride content of the blood for the accumulation of non-protein nitrogen must be stressed also in cardiac and renal diseases, patients suffering from which are kept for a long period on a salt-free diet. Besides the rôle of chlorides in the pathogenesis of acute extrarenal azotæmia, it is obvious that tissue proteins are broken down when the body weight is reducing rapidly (twenty kilograms in a few weeks in the case of the patient with duodenal ulcer). The sequence is probably as follows: a sudden great loss of fluid and salts occurs, a disturbance of mineral balance and of the general metabolism takes place, an abnormal protein breakdown occurs and there is an accumulation of nitrogen substances in the blood aggravating the initial vomiting and diarrhœa, thus creating a vicious circle.

Ambulatory Treatment of Pulmonary Tuberculosis by Artificial Pneumothorax.

J. ARTHUR MYERS (*The Journal of Thoracic Surgery*, December, 1933) has had considerable success with artificial pneumothorax in the treatment of tuberculous patients who were unable or unwilling to cease their ordinary avocation. He points out that this form of treatment was originally combined with a strict sanatorium routine or even with rest in bed for a period of from six to twelve months. Later, physicians became more liberal and allowed their patients to become ambulatory after a shorter time. In 1926 the author treated by artificial pneumothorax two patients who refused to become bed patients. In one collapse was maintained for sixteen months, in the other for three years, and both were in perfect health in 1932. The favourable outcome of these and other cases led him to lay down careful indications wherein collapse therapy could be considered without the additional rest in bed which this usually implies. A favourable type of patient is the adolescent or person in the early twenties whose general condition is good and who has a minimal or moderately advanced unilateral progressive lesion. The author has treated thirty-four patients by ambulatory artificial pneumothorax, the majority continuing their work without intermission. In all, satisfactory collapse was obtained, and all at the moment the author wrote were carrying on their usual work. Of these, nine had received the treatment for more than two years, and another fifteen for more than one year. The author found that complications, such as effusion, did not occur any more frequently in these than in resting patients. He regards this treatment as justified in carefully selected cases, particularly because it reduces the bed requirement for the community and thus aids in the isolation of carriers of tubercle bacilli whose disease cannot be treated successfully.

Latent Icterus in Chronic Cholecystitis.

ÉTIENNE CHABROL AND ANDRÉ BUSSON (*La Presse Médicale*, February 10, 1934) write of the value of blood bile estimations in the diagnosis and treatment of chronic cholecystitis, and especially of the ill-defined condition biliary dyspepsia. Bilirubin may amount to 20 milligrammes per litre of blood without constituting hypercholæmia. In 45 of their patients, several weeks after a "painful crisis" and without any clinical jaundice, the bilirubin per litre of blood was found to be: between 45 and 50 milligrammes, 6 cases; 40 to 45 milligrammes, 14 cases; 35 to 40 milligrammes, 7 cases; 30 to 35 milligrammes, 4 cases; 25 to 30 milligrammes, 7 cases; 20 to 25 milligrammes, 1 case; below 20 milligrammes, 6 cases. In diagnosis between cholecystitis without jaundice and gastric or duodenal ulcer such estimations are valuable, and also as a guide to treatment. The success or otherwise of non-operative treatment of cholecystitis may be gauged by the bilirubin of the blood. If this has fallen to 16 milligrammes or lower, operation may be deferred; if, on the other hand, after three months of rest and strict diet, the bilirubin remains at 30 milligrammes or higher, operation is indicated. General anæsthesia did not produce a bilirubin rise in cases of appendicectomy, herniotomy, hysterectomy, and oophorectomy investigated by the authors. After cholecystectomy the blood bilirubin fell or rose according to whether the bile duct was drained or not. Usually the rise did not last more than a week or so. When drainage is adopted, if the blood bilirubin falls and remains normal, the surgeon may feel justified in removing the tube and allowing the wound to close.

Fatal Tularæmia.

IN reporting the history and autopsy findings in a fatal case of tularæmia, Lewis P. Gundry and C. Gardner Warner (*Annals of Internal Medicine*, January, 1934) review the literature on fourteen recorded cases, discussing both the clinical and pathological findings in these instances. The pulmonary lesions are prominent in the visceral pathology, and the outstanding features may be summarized as follows: The process is a confluent, lobular pneumonia, with marked involvement of the interstitial tissues; the exudates contain a predominant number of lymphocytic cells; vascular lesions are found characterized by subintimal œdema, often associated with thrombosis. These vascular occlusions offer the best explanation of the areas of necrosis which are typical features of tularæmic pathology.

Special Abstract.

THE ANÆMIAS OF INFANCY AND EARLY CHILDHOOD.

As their contribution to the general interest recently aroused in the subject of the anæmias, the editors of *Archives of Disease in Childhood* arranged for a series of authoritative articles on the anæmias of childhood to appear in five successive issues of their journal between April and December, 1933. Simultaneously an opportunity was afforded the workers at the Children's Hospital and Department of Diseases of Children of the University, Birmingham, to make known the results of their three years' investigation of this subject. Taken together, the contributions form a notable chapter in the English literature dealing with diseases of the blood; they plainly express the most modern viewpoints and embody a considerable amount of original work and thought. The series is divided into nine parts as follows:

Part I. Introduction.

The subject is introduced by Professor L. G. Parsons, of Birmingham, the general sponsor for the series. He describes the last decade as the renaissance of hæmatology and pays a tribute to the previous work of Helen Mackay in England, and to Hart, Steenbock, Elvehjem and Waddell in America. The necessity for the appreciation of the word "erythron" as connoting the blood cells, serum and bone marrow considered together as a deliberate tissue or organ is vitally essential to the latter day conception of the anæmias. Describing the erythron in early childhood, Professor Parsons draws attention to the wide extent and activity of the red marrow, allowing in emergency little opportunity of further production, so that at such times extramedullary centres of hæmatopoiesis may be required. No yellow marrow or macroscopic fat is visible in the bones until the age of twelve or fourteen years. These fatty changes occur more rapidly in the long bones of the lower extremity than in those of the upper. (The factors concerned in the maturation of the erythrocyte, as outlined by Witt, Strauss and Castle, are then described.) The author then describes the changes in character, numbers and hæmoglobin content during the first week of extrauterine life. He refers to work of Van Creveld on the Price-Jones curves of infants during the first few weeks of life, both premature and full-term, in which the essential difference lay in a shift to the left in the curve of the premature infant during the third and following weeks after birth. The bilirubin circulation is described, as well as the mechanism of physiological *icterus neonatorum*. Goldbloom and Gotlieb had shown that the fragility of the red cells directly after birth is greater than normal and that *icterus neonatorum* is due to this factor and to the change from the anoxæmia of intrauterine life to conditions in which the oxygen supply is much greater. Severe and unchecked hæmolysis occurs in familial *icterus gravis*. The fall in hæmoglobin after birth is more prolonged than that of the concentration of erythrocytes. By reference to "normal" curves constructed by Helen Mackay it is possible to classify infants as short of circulating hæmoglobin or not, according to their age and diet. Early hæmolysis has been accepted as the cause of the post-natal fall in hæmoglobin, but Josephs suspected deficient production as a further factor, since, in the early period, iron administration has no influence on reticulocytosis or on the falling rate of hæmoglobin.

The majority of anæmias of infancy may be classified into two groups: (*a*) those in which there is a deficiency in one or more of the elements necessary for the synthesis of hæmoglobin or for the production of the erythrocyte or both; (*b*) those in which there is increased destruction (deficiency anæmias) of the erythronic elements (hæmolytic or erythronoclastic anæmias, which embodies a brand new addition to the nosology of the subject). It is frequently very difficult in infancy to determine in any particular case whether the anæmia arises solely as a deficiency disease or as a result of destruction of some part of the erythron, or, as in the case of infections, from both.

Parsons and his colleagues consider von Jaksch's anæmia as a hæmolytic anæmia. Primary anæmia of the newborn is subdivided into hæmolytic anæmia and deficiency anæmia due to defective antenatal storage. Aplastic anæmia is said to be extremely rare in infancy, of undetermined ætiology, but favoured by this series towards a primary destructive disease of the marrow series. Sickle-celled anæmia and acholuric jaundice are regarded as congenital malformations of the erythron. Goat's milk anæmia and the anæmias of leuchæmia are included in review.

Part II. The Effect of Yeast on Nutritional Anæmia of Rats.

Professor Parsons is associated with Dr. E. M. Hickmans in the work on the effect of yeast on nutritional anæmia of rats. They commence by an abbreviated review of the formidable literature on nutritional anæmia. Experiments were repeated which had shown that yeast had a curative effect on a nutritional anæmia of the rat, readily produced in two months by feeding the animals exclusively on liquid milk. The remainder of the investigation centred around the question as to which elements of the yeast were most beneficial. After cure by yeast the animals were much more fertile than those control rats cured by copper and iron, although the progeny was found to be anæmic at the end of the lactation period (twenty-one days). Growth and reproduction were still abnormal when iron, copper, or both were added to the milk diet. The progeny of the rats fed on milk and yeast were examined three weeks after birth and showed a deficiency in iron, but not in copper. The percentage hæmoglobin of members of litters from rats fed on yeast and milk averaged 38. The values of different members of the same litter varied a good deal. The profound anæmia exhibited by the young rats when they were a week old was remarkable. The parent remained normal and her supply of iron did not suffer during and after gestation. The anæmic young rats were made use of for the testing of the curative agents recommended in nutritional anæmia. Yeast contains the vitamin *B* complex necessary for red cell maturation, iron, copper, manganese and a trace of arsenic. The proteins are zymocasein and cerevisine, containing mostly the amino-acids, glutamic acid, arginine and lysine. "Anæmic" yeast, a form prepared by special subculture, is free from inorganic iron or cytochrome, and total iron is one-third to one-half normal. Given to the anæmic rat progeny, it led to a cure, although more slowly than ordinary dry yeast. Additional iron permitted a more rapid hæmoglobin formation. The addition of yestamin also accelerated the cure. The curative effect of anæmic yeast could not have been wholly due to its hæmatin content, as compounds of hæmatin are not readily broken down in the alimentary canal, a fact which explains the failure of liver alone to cure nutritional anæmia. Iron, copper and yestamin in standard proportions produced an astonishingly good result. Certain results suggested that there was a shortage of vitamin *B* in anæmic yeast, but this showed only after feeding over two generations. Parsons and Hickmans proved also that there was insufficient iron in the milk to cure the anæmia. One gramme of yeast extract (containing the vitamin *B* complex, but no hæmatin) will cure. "Marmite" was found to convey these factors best. Experiments to determine whether vitamin B_1 or B_2 is the most effective in cure are already in progress. The difficulty in assessing the part of other factors, iron, copper and amino-acids, has been rendered more difficult by the discrepancies between quantitative and spectroscopic estimation. Apparently there is inadequate assimilable iron in the mixture. Copper is merely a catalyst to iron, so the good effect must be largely due to amino-acids or vitamin *B* or both. Unaided, the vitamin *B* complex is not enough. The amino-acids are usually thought to be ineffective. No direct work was undertaken with them in this investigation. The authors state that the complete picture has so far eluded them.

Part III. The Anhæmopoietic Anæmias, Nutritional Anæmia, and Anæmias of Prematurity, Scurvy and Cœliac Disease.

Professor Parsons and J. C. Hawksley undertake Part III.

Helen Mackay has demonstrated that nutritional anæmia of a mild degree is almost universal in breast and artificially fed babies; this is a simple iron deficiency, except in the presence of an infection. Nutritional anæmia of this type is due to deficient storage of iron in intrauterine life, or from an inadequate supply of iron in the diet, or both. Copper is stored in like manner. Only iron and copper are stored in this way and reach their peak at birth. There may be a slight further post-natal rise after birth due to hæmolysis (Sheldon). (Congenital nutritional anæmia differs from nutritional anæmia of later infancy only in the fact that the child may come under observation shortly after birth, or there is a pallor from birth.) It is essential in diagnosis to exclude an abnormal hæmolysis. Nutritional anæmia is microcytic and hypochromic, without evidence of marrow response until iron or other missing factors are supplied, when reticulocytosis occurs. The authors are satisfied that an adequate maternal diet will prevent nutritional anæmia. Certain mothers have power to retain their iron to the detriment of the fœtus, a state of affairs entirely different from that which obtains with calcium. The only clinical abnormality detectable in the child is the pallor. For this to have become noticeable the hæmoglobin must have fallen below 60%. There is no icteric component. Bronchitis or otorrhœa is frequently present. The authors draw attention to an undue mental backwardness. There is no evidence that a concurrent rickets has any relationship to the anæmia, except that both are nutritional in origin. The white cells do not show any constant quantitative or qualitative alteration. Any splenomegaly is more probably related to infection. The authors gave their patients reduced iron in doses of one-half to one grain thrice daily. If the cause has been an iron lack, reticulocytosis and a rise to a supranormal concentration of erythrocytes follow, the hæmoglobin value rising more slowly. If the response is only partial, other factors besides yeast and orange juice are added. The dependence of reduced iron on the presence of adequate gastric acidity is referred to, and a hypoacidity is suggested as a possible reason for inadequate response. The addition of copper in the form of one cubic centimetre of a 0·5% solution of copper sulphate per kilogram of body weight given daily in milk was further found beneficial. The effect of copper seemed to be solely on the hæmoglobin value, not on the concentration of erythrocytes. Copper is not necessary for iron assimilation, but is necessary for the synthesis of hæmoglobin. The authors have attempted further to answer the question: "Can the administration of a pure iron salt cure nutritional anæmia in the infant, or is copper also invariably necessary?" Chemically pure ferrous sulphate was used, twelve grains daily, in glucose to prevent oxidation, and the results were more rapid than those that Mackay had obtained with the scale preparations. A modification of ferrous iron, called "Ferofax", has been since produced by British Colloids Limited as a result of this work. One case of its successful use is reported in which there was no possible source of copper other than the very slight impurity of this character in medicinal glucose, and that perhaps stored antenatally. The results with dried yeast alone, in the form of yestamin, were disappointing when from one-quarter to four drachms were used; not even a "reticulocyte kick" resulted. No conclusion was reached as to whether yeast and iron together produced a quicker result than iron alone. It is obvious that yeast is much less efficacious in the treatment of nutritional anæmia in the child than in the rat. In neglected cases natural cure of nutritional anæmia follows weaning, when the child begins to take a mixed dietary.

The ætiology and natural history of the so-called physiological anæmia of prematurity are next discussed. The blood of the premature infant behaves like that of a full-term child up till the fourth week and then shows a marked drop in hæmoglobin and red cells, reaching a minimum about the eighth week; it reaches normal again only about the sixth or eighth month. The authors defend the hypothesis of antenatal shortage of iron as the cause. Though the administration of iron will not prevent the fall of hæmoglobin content, it will cure resulting anæmia. The mechanism of this fall, whether it is due to lessened blood formation or increased blood destruction, has nothing to do with the adequacy of antenatal iron storage. Clinically the anæmia of prematurity resembles nutritional anæmia, except that splenomegaly and lymphatic hyperplasia are more common, and in the early stage the anæmia is of a hyperchromic type. Severe grades of anæmia of prematurity may be prevented by the administration of iron and dried liver together, or reduced iron and vitamin *D*. Blood transfusions are similarly effective, and serum may be administered for *icterus gravis neonatorum*.

Anæmia occurs in scurvy, when hæmatopoiesis does not occur after a hæmorrhage, except in the presence of vitamin *C*. Copper should not be given with vitamin *C*, which it destroys.

Anæmia in cœliac disease may be either hypochromic or, less commonly, hyperchromic. The former is essentially a nutritional anæmia due to lack of iron, copper and vitamins. The hyperchromic variety is in line with that seen in sprue, gastro-colic fistula, tropical anæmia *et cetera*. It differs from true pernicious anæmia in that free hydrochloric acid is frequently present in the stomach, there is a striking absence of poikilocytosis, and the anæmia responds to "Marmite" (extrinsic factor); also the indirect Van den Bergh reaction is low in unitage. In children, however, hyperchromic anæmia is of the greatest rarity. Two conditions are suggested as necessary for its development: (*a*) long-standing cœliac disease, and (*b*) previous inadequate treatment, probably inadequate vitamin B_2. An erythroblastic form of anæmia, resembling Cooley's anæmia clinically and hæmatologically, but responding to iron, has also been described. The authors review nineteen cases of cœliac disease of their own. Nine patients were free from anæmia, six showed anæmia of a hypochromic type, three a mild hyperchromic anæmia, and one hyperchromic macrocytic anæmia. Ferrous sulphate in adequate doses (twelve grammes daily) never failed to cure the hypochromic anæmia. The mild degrees of hyperchromic anæmia remained unbenefited by "Marmite" or desiccated hog's stomach, duodenum or jejunum, except in one case. It is suggested that the cœliac disease in this case, and perhaps in all children, is associated with a defect in Castle's intrinsic factor, as in sprue. True pernicious anæmia and bothriocephalus anæmia are stated practically never to occur in the first decade of life.

Part IV. The Hæmolytic (Erythronoclastic) Anæmias of the Neonatal Period.

Parsons, Hawksley and Gittins combine in a description of the hæmolytic (erythronoclastic) anæmias of the neonatal period, with special reference to erythroblastosis of the newborn. They prefer the term "erythronoclastic" because it embraces interference with or destruction of the hæmopoietic centres, together with "hæmolysis" of the circulating cells. Such anæmias are hyperchromic with marked anisocytosis and poikilocytosis. Megaloblasts and normoblasts appear from over-activity of the marrow and not from a maturation deficiency. The first group to be discussed is the hæmolytic anæmia of the newborn. This may or may not be associated with all degrees of *hydrops fœtalis* and/or jaundice. Severe cases of *hydrops fœtalis* are usually seen in stillborn infants. Fluid is found in all serous cavities, with subcutaneous dropsy and enlargement of the liver and spleen. The blood shows a severe anæmia, with an erythroblastæmia or increase in nucleated red cells varying from 5,000 to 297,000 per cubic millimetre. The white cell precursors are also sometimes found in great numbers. The œdema is regarded as secondary to the anæmia, but this may cause the heart to hypertrophy and later to fail. These authors report one case. *Erythroblastosis neonatorum* forms one variety of *icterus gravis*, which is frequently familial. Jaundice begins just after birth and deepens rapidly until death

occurs about the fourth or fifth day. There is frequently slight enlargement of the liver, more definitely the spleen. The urine shows urobilinogen, urobilin in excess, and bile pigments, but the stools are always acholic. A typical sufferer had 2,950,000 red cells per cubic millimetre, of which 213,300 per cubic millimetre were nucleated. Cases have been described in which no abnormality of red or white cells was apparent. Blood taken as the jaundice is fading reveals a profound hyperchromic anæmia with considerable polychromasia and anisocytosis and an eosinophilia during recovery. No subsequent treatment is required as, if the child survives, spontaneous recovery occurs; but Hampson's method, which consists in the intramuscular injection of ten cubic centimetres of parental blood serum daily is sometimes very successful if commenced on the first day. Serum is preferable, the authors contend, to whole blood. Early diagnosis may be suggested by the yellow colour of the *vernix caseosa* in erythroblastosis of the newborn. Odd examples of hæmolytic anæmia later in the neonatal period without œdema or *icterus gravis* are described.

The authors have examined material from four fatal instances of hæmolytic anæmia of the newborn. Macroscopically all organs were deeply bile-stained. No staining of the basal ganglia was observed, as has been described. The liver was enlarged, but not cirrhotic. The bone marrow was a rich red throughout. Histologically the most striking feature was the embryonic blood formation in the hepatic sinusoids, producing a picture similar to a leuchæmic process, but consisting of cells 8μ to 10μ long, with vesicular rounded nuclei 7μ in size. The staining in these proerythroblasts in general was denser than that of myeloblasts or premyelocytes. Closely associated with these cells were large numbers of erythroblasts themselves. There was a considerable amount of hæmosiderin present, but no toxic degeneration of the liver cells themselves. Erythropoiesis was also present in the spleen, but more variable from case to case. The marrow activity was mainly confined to the myeloid side and normoblasts were scanty. It must be remembered that some degree of extramedullary hæmopoiesis is common in healthy newborn children, and sections from a premature infant of eight months usually show extensive erythropoiesis in the liver.

Part V. The Hæmolytic (Erythronoclastic) Anæmias of Later Infancy and Childhood.

The hæmolytic (erythronoclastic) anæmias of later infancy and childhood are described by Professor Parsons and Dr. J. C. Hawksley. They divide their patients into four groups as follows:

1. *Acute Hæmolytic Anæmia (Lederer Type).*—These children are suddenly afflicted by a severe hyperchromic anæmia, enlargement of liver and spleen, and an intense marrow reaction in the form of leucocytosis. All make a rapid and spontaneous recovery. The present authors have not observed the gastro-intestinal irritation described in Lederer's original series, but agree that an infectious origin is probable. Thirty-six cases in all have been described, in which eleven patients were between the ages of two and eleven years. Some patients recover from the hæmolytic anæmia but continue to manifest a nutritional anæmia. The intercurrence of an infection may delay recovery. A blood transfusion may hasten a slow improvement.

2. *Subacute Hæmolytic Anæmia.*—Subacute hæmolytic anæmia is merely the less dramatic variant of the above with a more gradual onset and a tendency in some cases towards definite chronicity. Parsons and Hawksley state that a diagnosis of acute or subacute hæmolytic anæmia should always be viewed with suspicion unless the patient recovers or the diagnosis is confirmed at autopsy, so great at times is the difficulty of separating these from leuchæmia.

3. *Von Jaksch's Syndrome (Subchronic Hæmolytic Anæmia).*—The writers refer to the traditional British description of von Jaksch's syndrome, that is, occurring only under three years of age and causing a waxy pallor, hepatomegaly and a large splenomegaly, œdema and petechiæ, but no enlargement of lymph glands or interference with general nutrition. American observers, led by Blackfan, Baty and Diamond, regard it as an anæmia with splenomegaly, leucocytosis and a moderate number of immature leucocytes and erythrocytes in the blood. The blood picture of the English cases shows a hypochromic anæmia, a leucocytosis with myelocytes in greater number than in any other condition except leuchæmia, for example, 2% to 6%. These patients are nowadays rather a rarity. Neither English nor American descriptions fit in exactly with that of von Jaksch, who remarked on the absence of eosinophile cells and the swelling of lymph glands. Parsons and Hawksley do not consider rickets as part of the von Jaksch syndrome. Apparently a good description of the disease was furnished in 1891 by Luzet, a pupil of Hayem, while Cooley, who described the disease recently in America, praises Eppinger's writings. Cooley has done good service in separating the erythroblastic anæmia of childhood from the motley variety of anæmias hitherto described as von Jaksch's anæmia. This is a hypochromic anæmia with an accompanying leucocytosis and an unusual number of nucleated red cells. Splenomegaly is very marked, there is an icteric tinge to the skin, a mongoloid facies and characteristic skull radiograms. The affection is limited almost entirely to Syrian, Greek or Italian stock. The prognosis is grave, though splenectomy causes some abatement, coincident with a sustained rise in normoblasts. Parsons and Hawksley conclude by advising the abandonment of the term von Jaksch's anæmia, and regard the British description as really referring to a rather more chronic form of the group of subacute hæmolytic anæmias referred to above. They quote examples of von Jaksch's anæmia becoming converted into an acute leuchæmia on the one hand and, as in the case of the other hæmolytic anæmias, into a nutritional anæmia. They can offer no new views as to ætiology. Rickets and syphilis are denied a place. They consider splenectomy too drastic, in view of the ultimately good prognosis.

4. *Other Forms of Hæmolytic Anæmia.*—Among other forms of anæmia are: (*a*) sickle-celled anæmia, confined entirely to negroes, and (*b*) acholuric familial jaundice. Both are probably congenital malformations of the erythron. The latter probably represents the most chronic hæmolytic anæmia, lying at one extreme of the scale. Differences to be noted are the fragility of the cells and the higher packed red cell volume (more globular red cells) in acholuric jaundice. Case histories of nine patients showing hæmolytic anæmia of later childhood are included.

Part VI. Nutritional Anæmia in Mother and Child.

A. V. Neale and J. C. Hawksley continue in a discussion of the blood changes in the mother during pregnancy as a result of the drain on her hæmopoietic resources by the child. Together with a steady call on the mateal iron reserve, there is a temporary anacidity or hypoacidity with a lack of the intrinsic factor of Castle, while further embarrassment may be caused by a deficiency in external factors in the diet consumed. All or only some of these causes may be operative, but the anæmia, whether hypochromic or hyperchromic, has far-reaching effects upon the *fœtus in utero*. A congenital anæmia may thus be a true nutritional anæmia. Fœtal storage of iron increases rapidly during the last weeks of pregnancy to provide for the milk-feeding period of early post-natal life.

According to the majority of observers, infants born of anæmic mothers have normal blood pictures at birth. Neale and Hawksley dispute this conception, at least in part, as a result of observing the blood of young rats born of mothers rendered nutritionally anæmic. They admit that an infant born of an anæmic mother is not necessarily anæmic. The striking difference in this connexion between iron and calcium is that the mother must surrender her calcium, even if she acquires osteomalacia, whereas the mother can and sometimes does retain iron to the detriment of the fœtus. Using Mackay's standard of normal hæmoglobin for an infant as 140%, Neale and

Hawksley can detect anæmic children in the series of Strauss and Castle. The authors discuss their cases of nutritional anæmia in mother and/or infant under the headings of anæmia in twin births and anæmia in single births. All possible permutations and combinations have been encountered. The division between twins of a subnormal or even just normal transplacental supply of iron is an adequate explanation of their hypochromic anæmia. Prematurity whereby the infant loses the specially abundant supply of iron during the last month of gestation, is a fruitful cause of congenital nutritional anæmia or that developing at a later date. The writers record instances of one normal and one anæmic twin born of a normal mother, frequently combined with a low body weight in the child affected. A remarkable example of a healthy, good-coloured baby born of a severely anæmic mother is recounted. Anæmia was present before the pregnancy and was aggravated by its presence, but rapidly responded to iron and yeast after labour. Neale and Hawksley regard this series as human corroboration of the facts observed in nutritional anæmia in rats, described above, where adequate iron and yeast not only insured a cure of the animals affected, but allowed them to propagate and produce a litter with a supranormal hæmoglobin value.

Part VII. Monocytic Reaction in Myelosis.

Robert Gittins and J. C. Hawksley record a case of monocytic leuchæmia in a child of almost three years of age, whose blood picture had been consistently followed for a period of six months. The monocytes (or histiocytes), which are a form of miniature myelocyte, show a rounded or oval nucleus with a typical streaky appearance of the cytoplasm after staining with Leishmann's stain and failure to obtain an oxydase reaction. They varied between 29% and 42% of the total white cells. The case report is illustrated by admirable drawings and photographs of these cells. No increase of monocytes was found anywhere in the tissues, so no light was thrown on the origin of these cells. Characteristically monocytic leuchæmia shows: (*a*) an increase in the peripheral blood of monocytes; and (*b*) a cellular proliferation of reticulo-endothelial cells in the organs containing this system. Accompanying this proliferation is a widespread infiltration with cells of monocytic character. Cellular areas of myeloid metaplasia may also be found, and in this particular instance the final blood and *post mortem* appearances were those of myelosis (myeloid leuchæmia). The authors had already reported elsewhere a case of pure monocytic leuchæmia and have decided that the reticulo-endothelium of the lymph gland takes a large share in the production of these cells. They think that monocytosis would be found more frequently if chronic leuchæmia were a commoner occurrence in childhood. The inference is that in the presence of monocytosis an underlying myelosis should be borne in mind. An immaturity of granulocytes cannot be relied upon to reveal itself in the early stages of an illness that ultimately proves itself to be leuchæmic; the distinction between these two conditions may therefore be impossible to make. Infective mononucleosis shows no immaturity in the white cells or anæmia, and there is not the clinical deterioration of the condition under discussion. The eosinophile count may help. An entire absence of eosinophile cells is reported as common in monocytic leuchæmia or leuchæmic reticulo-endotheliosis.

Part VIII. Leuchæmia (Leucosis) in Children.

Robert Gittins contributes an excellent monograph on leuchæmia (leucosis) in children; it is based on a personal study of fourteen cases and is most adequately illustrated. Chronic leuchæmia in childhood is extremely rare, and this series was composed all of acute cases. Too much emphasis can scarcely be laid on the acuteness and on the aleuchæmic character of the disease in childhood. Erythronoclastic and von Jaksch's anæmia, infective mononucleosis, some infective septic and nutritional anæmias may also call forth immature leucocytes and become confused with true leuchæmia. Gittins prefers the term leucosis to leuchæmia, in childhood at least, as the underlying process of disease is one affecting the leucoblastic tissues rather than the leucocytes. He divides leucosis into two chief forms, myelosis and lymphadenosis, and each of these into leuchæmic, aleucocythæmic and aleuchæmic varieties, which are self-explanatory. The commonest type, as already stated, is the aleucocythæmic lymphadenosis.

In an account of the symptomatology he refers to the commonest age period as ten months to twelve years, to the insidious onset, the waxy pallor and the greater incidence of splenomegaly in myelosis than lymphadenosis. Gingivitis, stomatitis and pharyngitis were not features of Gittins's series. Hæmorrhagic retinitis is surprisingly rare, but petechiæ and purpura are common in the skin. Bone changes are a rare manifestation in childhood, but osteoporosis, cortical thinning and elevation of the periosteum have been observed (Karshner). Fever of an irregular type is usually present. Rarely the nervous system is infiltrated with appropriate localizing features. The course of this disease is short, usually under two months. The degree of enlargement of the liver, spleen and lymph glands is quite disproportional to the rapidity of the course. The rare chronic leucosis of childhood is almost always a myelosis.

With regard to the hæmatology, there is always a very marked hypochromic anæmia with a great reduction in platelets. Red cell volume is small, for example, an index of 0·74. Degenerative changes and immaturity of red cells are commoner in myelosis than in lymphadenosis. Indeed, the presence of nucleated red cells in any number should make one suspect a hæmolytic anæmia.

A point of very great therapeutic importance in lymphadenosis is that the proportion of lymphocytes and lymphoblasts is high, up to 98·7%, and that all other types of cells are absolutely reduced. The variation in the differential count in myelosis is much wider, including the eosinophile and basophile cells and their precursors. Monocytes are usually low in numbers in either variety of leucosis. Naegeli's oxydase reaction is of value, as if it is positive, it places a doubtful cell in the granular class, except in the case of the most immature myeloblast. The differentiation between lymphoblast and myeloblast may still be difficult at times, but can nearly always be made after architectural study. As regards diagnosis, the possibility of irritation of the bone marrow by foreign (for example, tumour, lymphadenoma) cells must be borne in mind; but careful study, writes Gittins, always reveals a very pure type of marrow reaction, myelotic or lymphadenotic; that is, "mixed" types of leucocis probably do not exist. The marrow apparently, in the early stages of lymphadenosis, however, sometimes responds to the "foreign" invasion by the putting forth of all types of immature leucocytes; but this stage is usually over by the time the child comes under medical observation, that is, aphasia of the granular leucon has occurred and the leuchæmic picture has become pure. In addition to the other diagnostic possibilities mentioned above, difficulty may rarely be encountered in distinguishing leucosis from aplastic anæmia. Here the maturity of the white cells in the latter condition is sufficient guide, together with the presence of leucopenia. *Osteosclerosis fragilis generalisata* (Albers-Schönberg's disease) is mentioned by Gittins as usually showing blood changes of a leuchæmic type, and he reports such a patient. The reticulo-endothelioses have an entirely different blood, if not clinical, picture. Lymphosarcoma simulates lymphadenosis in many ways, and the process seems to differ only in the lesions being more focal in distribution.

A good description follows of staining methods and the histological features of blood and tissue films in each variety of leucosis, also a discussion on the differentiation of myelopoietic from erythropoietic foci. Reference is made to the activity seen in the Kupffer and other cells of the reticulo-endothelial system. Gittins states that the histiocyte probably arises from the reticulo-endothelium. They appeared in numbers in the hepatic sinusoids of his patients.

Under the title "Histopathology of Leukæmia" Gittins describes the architecture of leuchæmic tissues in both forms of leucosis in the organs affected. He has no sug-

gestions to offer regarding treatment. Liver and Fowler's solution were used without effect in the last example of his series. His views on ætiology are based on a firm impression that leucosis is a neoplastic process. Leucocytosis and leucosis appear to be respectively hyperplastic and neoplastic phenomena analogous to reparative processes and malignant focal newgrowths. He discusses the infective theory as most unlikely, as leucosis is not initiated by any known process of infectious disease in man, and isolated instances of infectious processes in the course of leuchæmia are not convincing.

Part IX. Anæmia and Reticulo-Endotheliosis.

Robert Gittins continues with a contribution on the subject of anæmia and reticulo-endotheliosis, which has hitherto received scant attention at the hands of both clinician and pathologist. His description is based upon four examples of hæmolytic anæmia which showed outstanding activity of the reticulo-endothelial system, a response, he thinks, to damage of this organ. These patients are described in detail, particularly the histological appearances of spleen, liver, marrow and lymph glands. The striking feature of all these preparations was the swelling and liberation of endothelial cells lining sinusoids and the presence in abundant quantity of phagocytic histiocytes. The reticulum in each instance was swollen and somewhat proliferated, as shown by the Bielchowsky silver method. Hæmosiderin was readily demonstrated in all tissues by the Turnbull-Huecke method. Individually the four patients, who were all under the age of two years (one aged seventeen months) varied considerably in their clinical aspects and in the degree of activity shown by the reticulo-endothelial system. In one child areas of erythropoiesis were to be seen in liver and spleen. Gittins's thesis, however, is that the common changes in the reticulo-endothelial system were reactive in nature. Thus the first child was at the onset diagnosed clinically as furnishing an example of subacute erythronoclastic (hæmolytic) anæmia); then, owing to the severity of the erythronoclastic process, as aplastic anæmia; later as von Jaksch's anæmia, followed by a return of the aplastic state. On the histological side the prominent feature was the production, apparently from reticulum cells, of quantities of free wandering histiocytes or macrophages, which were busily engaged in scavenging red cells. Gittins holds that this activity is consequent on and not productive of the erythronoclastic anæmia. The large numbers of reticular fibrils present in the final and most chronic case suggested that this is the ultimate change in the reticulum; and the aplastic state may then be related to such a change in the hæmopoietic centres. It is apparent that if the patient is seen in the stage of erythrocyte damage only, the blood picture may be that of regeneration. Should the bone marrow also suffer heavily, the blood picture will naturally be aplastic or anhæmopoietic. Another possible factor is the existence or not of embryonic hæmopoietic foci at the time of commencement of the erythronoclastic process. Finally, if the erythronoclasis is mild but prolonged, reticulo-endothelial proliferation will replace hæmopoietic foci wherever they be and contribute further to the state of anhæmopoiesis. These possibilities and their variable degree of development may account for the variety of blood pictures found in the so-called von Jaksch's anæmia, which is regarded by Gittins and his co-workers as a subchronic erythronoclastic anæmia.

Gittins next proceeds to give an historical and physiological description of the reticulo-endothelial system with reference to its scavenging and immunological duties. He supports Dameshek and Kiyono's view that both monocytes and histiocytes are produced by the fixed cells of the system. The histiocytes are soon filtered out of the circulation. Those seen in the peripheral blood as monocytes may have only just been produced.

The next remarks concern the question of reticulo-endotheliosis. A brief summary is given of the German case reports under this title by Lederer, Schultz, Akiba *et cetera*, and of leuchæmic reticulo-endotheliosis as exemplified earlier in this series. It is reasonable to expect that aleuchæmic forms or phases occur. Most of the patients so far reported are apparently non-leucotic but reactive to some kind of infection.

Gittins classifies the reactions of the reticulo-endothelial system in disease as follows:

(*A*) Reactive:
- (i) To blood destruction.
- (ii) To sepsis and infection.
  - (*a*) Glandular fever: reticulo-endotheliosis of lymph glands.
  - (*b*) Typhoid fever.
  - (*c*) Infection.
  - (*d*) Granulomatosis, for example, Hodgkin's disease *et cetera*.
- (iii) To chemical abnormalities: Gaucher's disease *et cetera*.

(*B*) Focal neoplastic:
Reticuloma.
Hæmendothelioma.

(*C*) Leucotic: reticulo-endothelial leucosis: leuchæmic or aleuchæmic reticulo-endotheliosis or monocytic or histiocytic leuchæmia.

The term reticulo-endotheliosis has then to serve for conditions which may differ in nature as widely as reactive hyperplasia and neoplasia. A further alternative in nomenclature for leucotic reticulo-endotheliosis is "reticulo-endothelial leucosis" to contrast with myeloid and lymphatic leucosis. As regards the disputed question of the genealogy of white cells, Gittins claims that a study of his case of monocytic leuchæmia supports the polyphyletic theory rather than the monophyletic, as it would be expected that such a leucosis would show cytological features less distinctive of histiocytes and a tendency to produce rather a myelosis or a lymphadenosis.

The reticulo-endothelial system in post-natal life cannot be regarded as a progenitor of white blood cells. The appearances of the fixed cells of the reticulo-endothelial system, when swollen, have in the past deceived observers, making them believe that they are witnessing transition forms between cells of the lymphoblast and myeloblast class. Gittins and his co-workers have found no evidence in their studies of an indifferent cell or hæmocytoblast, and the endothelial system has taken no part in the formation of myeloblasts, lymphoblasts and erythroblasts.

The description ends with a summary of the clinical features of the four patients first presented, as follows: (*a*) anæmia, (*b*) splenomegaly with slight enlargement of liver and lymph glands and a few petechiæ, the blood picture revealing an anæmia of moderate severity with a somewhat high colour index and signs of slight bone marrow regeneration, terminating by aplastic anæmia and hæmorrhagic features. Differential diagnosis is made from Banti's disease on the age question and that of hepatic cirrhosis, from acholuric jaundice, from Hodgkin's disease, from glandular fever and storage and leucotic forms of reticulo-endotheliosis, and from parasitic diseases like malaria and kala-azar. As regards treatment, the greatest hope lies in repeated blood transfusion when the anæmia is severe. Splenectomy has not been tried, and it would require very careful preparation for this hazardous operation.

This concludes the outline of a series of most careful and thoughtful papers on the modern aspects of pædiatric hæmatology. No conscientious student of this subject could afford to neglect a careful scrutiny of the original articles themselves.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE TASMANIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Tasmanian Museum, Hobart, on Tuesday, May 8, 1934, DR. W. E. L. H. CROWTHER, the President, in the chair.

Schoolboys and Rowing Races.

At the previous meeting of the Branch Dr. F. W. Fay and Dr. A. W. Shugg had been requested to draw up a report on the desirability of schoolboys under the age of seventeen years taking part in rowing races. They now presented their report as follows:

At the request of the Council, the undersigned beg to submit for the consideration of the Branch the following recommendations in respect of rowing for schoolboys:

(1) No boy should be permitted to row in the school boat race who is under the age of seventeen years, unless he produces a medical certificate of his fitness to compete in such race.

(2) That the distances should not be more than one mile.

These conclusions have been arrived at for the following reasons:

(1) The public schools boat races were commenced at a time when it was customary for certain boys to remain at school until they were twenty years of age. Twenty was the age limit for playing in any team.

(2) The crews were then manned by boys mostly eighteen and nineteen years of age. Now, however, boys leave school much earlier, and the boats are manned by boys whose ages are greatly below the ages of the boys for whom rowing was first introduced; the strain on the hearts of the younger boys is much greater than that on the older ones.

It is for these reasons that the above resolutions are put forward.

FRANK W. FAY.
A. W. SHUGG.

The report was adopted after the addition to Clause (1) of a sentence to the effect that such boys should also be under constant medical supervision during their course of training.

Society of Anæsthetists.

The meeting considered a letter that had been received from Dr. Geoffrey Kaye concerning the formation of an Australian society of anæsthetists. No action was taken in regard to it.

Rhinophyma.

DR. J. STODDART BARR read notes and showed stereoscopic photographs of a patient upon whom he had operated for rhinophyma. He explained that the condition had its origin in a seborrhœic dermatitis which led to hypernutrition and hypertrophy of the sebaceous glands. Treatment consisted in paring down the mass with a large, flat, sharp knife, the operator keeping a finger in the anterior nares as a guide to the amount of tissue to be removed.

DR. W. W. GIBLIN recalled a patient suffering from the same complaint whom he had seen in Saint Bartholomew's Hospital, London. The nose of this patient, who was a cabman, had become so enlarged that many prospective fares who hailed him had turned away after seeing his nose.

Dr. Barr was asked to discuss the regrowth of skin on the large raw area. He replied that this was rapid, since each divided sebaceous gland acted as a centre of epithelial growth.

A Macerated Twin.

DR. E. W. J. IRELAND showed and described the placenta of a patient who had been pregnant with uniovular twins, one of which was represented by a macerated fœtus. He said that after the normal delivery of a living child the placenta was duly voided and on examination was seen to present unusual features. The membrane on one side was seen to contain the flattened remains of a three-months fœtus. This presented the appearance of a bas-relief or a fossil in a rock. The amniotic cavity belonging to this twin was completely obliterated, the head could be seen as an opaque mass, probably partially calcified. The left arm, presumably, showed the presence of a well marked humerus; radius and ulna were just distinguishable; the lower limbs could just be traced by holding the specimen against the light. Dr. Ireland said that he reported the case as it must be a very rare occurrence. He had not seen or heard of any similar happening.

Pulmonary Tuberculosis.

Dr. Ireland also reported and showed a radiograph of a child who was suffering from pulmonary tuberculosis. He pointed out that the film showed the condition of the lungs of a young girl of eight years who had been suffering from somewhat unsatisfactory health for several years. This child was, in Dr. Ireland's opinion, exposed to tuberculous infection from infancy. Two aunts died of tuberculosis, one with meningitis, the second with pulmonary phthisis. The husband of the first mentioned was an inmate of the Tasmanian Sanatorium and died in his mother-in-law's home some months after leaving that institution. All these three lived more or less in the same home with the subject of the present discussion, and the uncle-in-law died there. The radiograph showed: (i) enlarged and calcareous glands around the hilus of both lungs; (ii) some small areas in the upper part of the left lung, probably calcified scars of healed tuberculosis; (iii) striated lines running away from the hilus in a radiating fashion, thickening and possibly fibrosis and calcification along the lines of bronchioles. These striations were, as usual, more marked in the lower lobes and showed up particularly well on the right side. Possibly thick fibrous adhesions to the diaphragm and between the pleural surface in other parts were continuations of these tracts.

On physical examination the lungs showed no marked signs, except that there was a definite imbalance in the inspiratory sounds on the two sides. The air intake on the left side was generally clear and forcible. That throughout the right was feeble and muffled, definitely deficient, even after coughing. This loss of inspiratory sound Dr. Ireland had in the past been attributing to narrowing of the trachea or bronchioles, through pressure of the enlarged glands in the hilus, but this was only a theory. The frequent experience of sanatorium medical officers, when first examining a patient after admission, was the almost complete lack of marked physical signs, with the exception of the vague lack of breath sounds in some areas or throughout one lung. After six weeks or two months in the institution a marked change occurred in most improving patients and the area or areas affected were most definitely indicated, he thought, by the occurrence of fibrosis in these areas. Sir Robert Philip, in his valuable clinics at the Victoria Dispensary, Edinburgh, had always insisted most strongly on the decided value of this imbalance or absolute lack of breath sound as an early indication of a tuberculous lesion in the lung. In the patient whose chest was depicted in the film shown by Dr. Ireland, treatment began soon to produce a marked diminution of this imbalance, probably, he thought, owing to the reduction of swelling and pressure at the right hilus.

It was true that there were numerous lesions which might cause deficient transmission of breath sounds, but he was referring to the lung sounds in this class of patient, where there was a strong suspicion of tuberculous affection of the lungs, but few clinical signs. These cases should always be referred to a competent radiologist, whose report should be of great value in deciding the diagnosis.

DR. F. SHORT, who had taken the radiograph, amplified Dr. Ireland's remarks about it. He said that the markings on the film were pathognomonic of phthisis. He stressed the importance of interpreting such films as a whole.

DR. T. C. BUTLER said that he had recently seen a man who seemed obviously ill. Just before coming to him the patient had been examined by a very competent physician, who had been able to discover no signs of disease. Dr. Butler also had been unable to elicit any abnormal physical signs, but in view of the patient's general condition he had sent him to a radiologist who had found signs of well established phthisis. Dr. Butler was beginning to place much greater reliance on X rays than on physical examination in the diagnosis of phthisis.

Epithelioma of the Anus.

DR. T. GIBLIN showed a specimen that he had removed that morning from a patient who was suffering from an epithelioma of the anus. He discussed the comparative value of surgery and radiation in the treatment of this condition.

DR. F. W. FAY observed that the inguinal glands had not yet been removed. He doubted, however, whether it was worth while removing them, since some of the lymphatics of the affected part probably ran to the internal iliac glands, which were not easily accessible.

DR. F. SHORT spoke of the advantages of the diathermy knife in the treatment of such conditions, and suggested that if deep X ray therapy were not available, semi-deep therapy might be of some value, since the lesion was comparatively superficial.

DR. E. BRETTINGHAM MOORE expressed the opinion that the ideal treatment of such a condition would include the application of deep X rays to the part before and after operation.

Post-Graduate Work.

ANNUAL REFRESHER COURSE IN MELBOURNE.

THE Melbourne Permanent Post-Graduate Committee has issued the following syllabus for the anual refresher course to be held in Melbourne from July 23 to August 3, 1934.

Monday, July 23, 1934.

9 to 9.30 a.m.—Melbourne Hospital: Registration at Post-Graduate Office, Melbourne Hospital.
9.30 to 11 a.m.—Melbourne Hospital: Dr. Eric L. Cooper, demonstration in pathology.
11.15 a.m. to 12.45 p.m.—Melbourne Hospital: Dr. C. H. Kellaway, the Weaver Bray effect—a demonstration—or "Music from the Cat's Point of View".
2.15 to 3.30 p.m.—Melbourne Hospital: Dr. S. O. Cowen, "The Treatment of Toxic Goitre".
3.30 to 5 p.m.—Melbourne Hospital: Dr. Julian Smith, Junior, operative demonstration of treatment of surgical conditions of the anus.
8.30 p.m.—Medical Society Hall: Professor David Barr, Lecture I, "The Functions of the Anterior Lobe of the Hypophysis".

Tuesday, July 24, 1934.

9.30 to 11 a.m.—Saint Vincent's Hospital: Dr. Frank Niall, "The Diagnosis and Treatment of Cardiac Failure".
11.15 a.m. to 12.45 p.m.—Saint Vincent's Hospital: Dr. H. B. Devine, "Operative Demonstrations of Simplified Technique in Routine Operations".
2.15 to 5 p.m.—University of Melbourne, Anatomy Department: Professor Wood-Jones and staff, demonstrations in surgical anatomy.
8.15 p.m.—Melbourne Hospital: Melbourne Hospital Clinical Society meeting.

Wednesday, July 25, 1934.

9.30 to 11 a.m.—Women's Hospital: Dr. W. D. Saltau, "Methods of and Indications for Induction of Labour".
11.15 a.m. to 12.45 p.m.—Women's Hospital: Dr. W. Ivon Hayes, "Indications for Cæsarean Section".
2.30 to 5 p.m.—Melbourne Hospital: Demonstration arranged by the Section of Anæsthesia.
8.30 p.m.—Medical Society Hall: Professor David Barr, Lecture II, "The Suprarenal Glands and Addison's Disease".

Thursday, July 26, 1934.

10 a.m. to 12.30 p.m.—Infectious Diseases Hospital, Fairfield: Dr. F. V. Scholes and staff, demonstrations of infectious diseases cases.
2.15 to 3.30 p.m.—Alfred Hospital: Dr. W. S. Newton, "The Management of the Chronic Pulmonary Cavity".
3.30 to 5 p.m.—Alfred Hospital: Dr. Fay Maclure, "Renal Colic".

Friday, July 27, 1934.

9.30 to 11 a.m.—Children's Hospital: Dr. Stewart Ferguson, "Dietetics and Diarrhœa in Infancy".
11.15 a.m. to 12.45 p.m.—Children's Hospital: Dr. Rupert Downes, "Intracranial Emergencies".
2.15 to 3.30 p.m.—Melbourne Hospital: Dr. R. P. McMeekin, "Peripheral Neuritis".
3.30 to 5 p.m.—Melbourne Hospital: Dr. Henry Searby, "Tumours in the Neck".
8.30 p.m.—Medical Society Hall: Professor David Barr, Lecture III, "Thyreotoxicosis".

Saturday, July 28, 1934.

9.30 a.m. to 12 noon.—Saint Vincent's Hospital: Dr. K. G. Colquhoun, "Skin Conditions met with in General Practice"; Dr. J. O'Sullivan, "The Radiological Examination of the Alimentary Tract and Chest".[1]

Monday, July 30, 1934.

9.30 to 11 a.m.—Children's Hospital: Dr. Robert Southby, "Symptoms and Treatment of Syphilis in Children".
11.15 a.m. to 12.45 p.m.—Children's Hospital: Dr. Douglas Stephens, "Limp in Infancy and Childhood".
2.15 to 3.30 p.m.—Melbourne Hospital: Dr. W. W. S. Johnston, "Some Aspects of Nephritis".
3.30 to 5 p.m.—Melbourne Hospital, Dr. W. G. D. Upjohn, "Bone Swellings".
8.30 p.m.—Medical Society Hall: Professor David Barr, Lecture IV, "Hyperinsulinism and Related Conditions".

Tuesday, July 31, 1934.

9.30 to 11 a.m.—Alfred Hospital: Dr. Ewen Downie, "The Management of Diabetes Mellitus".
11.15 a.m. to 12.45 p.m.—Alfred Hospital: Dr. C. H. Hembrow, "The Therapeutic Use of Shoes in Foot Complaints".
2.15 to 3.30 p.m.—Alfred Hospital: Dr. J. P. Major, "A Therapeutic Medley".
3.30 to 5 p.m.—Alfred Hospital: Dr. Balcombe Quick, "Methods of Skin Grafting".

Wednesday, August 1, 1934.

9.30 to 11 a.m.—Women's Hospital: Professor Marshall Allan, "Indications for Interference during Labour".
11.15 a.m. to 12.45 p.m.—Women's Hospital: Dr. B. Milne Sutherland, "The Treatment of Puerperal Sepsis".
8.30 p.m.—Medical Society Hall: Professor David Barr, Lecture V, "The Relation of the Parathyreoid Glands to Calcium Metabolism".

Thursday, August 2, 1934.

9.30 to 11 a.m.—Saint Vincent's Hospital: Dr. W. J. Newing, "Artificial Pneumothorax".
11.15 a.m. to 12.45 p.m.—Saint Vincent's Hospital: Dr. Gordon Shaw, "Surgical Infections of Joints".
2.15 to 5 p.m.—Eye and Ear Hospital: Ophthalmology—methods of examination, diagnosis and treatment of common diseases of the eye.
8.15 p.m.—Women's Hospital: Clinical meeting of the Victorian Branch of the British Medical Association.

Friday, August 3, 1934.

9.30 to 11.30 a.m.—College of Pharmacy: Dr. B. L. Stanton, "Pharmaceutical Problems associated with the 1932 Pharmacopœia".
2.15 to 3.30 p.m.—Melbourne Hospital: Dr. Leslie Hurley, "Practical Points in the Diagnosis and Treatment of the Anæmias".
3.30 to 5 p.m.—Melbourne Hospital: Dr. Hughes-Jones, Diverticulitis—a clinical demonstration.
8.30 p.m.—Medical Society Hall: Professor David Barr, Lecture VI, "Epidemic Encephalitis".

The lectures by Professor David Barr constitute a separate course; the fee for this course is four guineas.

Hospital Accommodation.

Arrangements have been made with the Committees of Management of the Melbourne and Alfred Hospitals

[1] These demonstrations will be divided into two sessions so that those desirous may change over at half time.

whereby a limited amount of accommodation will be available at these hospitals for those attending the course, at an additional fee of £3 3s. per week to cover board and lodging.

Applications should be made to the Honorary Secretary, Dr. W. W. S. Johnston, 61, Collins Street, Melbourne.

Special Subjects.

Those desirous of devoting time to special subjects are asked to communicate with the Honorary Secretary as soon as possible so that the necessary arrangements can be made.

Obituary.

FRANK NYULASY.

DR. FRANK NYULASY, who died at his residence, Toorak, Melbourne, on Monday, May 7, 1934, was born in Ballarat sixty-eight years ago. At an early age he went with his parents to New Zealand, where he was at school for some years; later, he returned with them to Australia. He went to the Melbourne Grammar School. Prior to entering the University of Melbourne law was chosen for him as a profession, but in a few months he decided that it held no appeal, and he gave up the idea. He entered the Medical School, where he graduated as Bachelor of Medicine and Bachelor of Surgery, and after that became house surgeon at the Melbourne Hospital. Here he worked under the late Sir Thomas Fitzgerald, from whom he learned much, particularly about surgical diagnosis.

At a dater period he wrote a thesis on polypoid endometritis. This was accepted for the higher degree of Doctor of Medicine; it was afterwards published by *The Journal of Obstetrics and Gynæcology of the British Empire*.

A paper read by Nyulasy at the Medical Congress in Adelaide, on "A Rare Form of Pelvic Tumour", attracted the attention of Professor Nekani, of the University of Budapest, Hungary, and Secretary of the Hungarian National Medical Association, who invited him to become a regular contributor to the official journal of that important body.

Nyulasy was for a number of years honorary surgeon at the Women's Hospital, Melbourne. Later he received the appointment from the Admiralty of surgeon and agent for the British Navy to His Majesty's ships in Australian waters.

In 1922 he went to London, where he met Sir Arthur Keith, President of the Royal Society of Medicine. After reading his paper on puerperal fever he was elected a fellow of the Society.

Nyulasy had many wide and varied interests. For three years he was President of the Royal Victorian Institute for the blind, but had to resign through lack of time to devote to this. He was one of the original members of the Big Brother Movement, and several of the boys he helped made good in Australia. He gave many lectures and addresses to different societies—the Royal Society of Saint George, the Shakespeare Society, the Dickens Fellowship, and others; some of these lectures were published.

Nyulasy was keenly interested in research work and has bequeathed a legacy of £1,000 to the Melbourne University in memory of his brother, the late Arthur Nyulasy.

Dr. W. J. Penfold writes:

I cannot let the death of Dr. Frank Nyulasy go by without writing a few lines expressive of my indebtedness to him for the friendship which he accorded me.

It was Dr. Nyulasy's habit, when any new bacteriological research appeared bearing on the subject of puerperal infections, to call at the Baker Institute and discuss the bearings of this research on obstetrical practice, and incidentally on the reputation of the obstetrician. He felt very keenly the reckless and ill-founded statements that were made reflecting on the practitioner, which practically made him the scapegoat for all the disastrous infections in the puerperium. The work of Colebrook and his colleagues at Queen Charlotte's was particularly interesting to him, and only recently he supplied a short abstract of that work to THE MEDICAL JOURNAL OF AUSTRALIA, which could not fail to be helpful.

Dr. Nyulasy was critical of new results and could put his case, in dealing with these results, in a lucid and able manner.

Having been at one time on the staff of the Women's Hospital, Melbourne, obstetrical and gynæcological subjects came to occupy a very predominant position in his professional life and judgement, and when the Baker Institute published an account of the infection of the fœtus arising *in utero* from the instrumental induction of labour he was keenly interested in the work.

When the institute was in financial and other difficulties his sympathy was extremely grateful.

In 1905 he was appointed surgeon and agent to the Royal Navy in Melbourne. The duties of this office

required him to attend sick sailors who were discharged from British warships because of their sickness, his relationship to these sailors being apparently not only that of surgeon, but a trustee of the Admiralty, having full charge of them. If they died, it was his duty to arrange for their burial, for the dispatch of their effects, and to forward his account to the Admiralty. Alternatively, if they recovered, he arranged for their dispatch to rejoin their ships. He was highly appreciative of England. He believed in her magnanimity, and had sympathy with her many difficulties.

Dr. Nyulasy had an artistic temperament and was very interested in good literature. He was a keen Shakespearean critic, and some of his published criticism received very favourable notice abroad from recognized authorities. He lectured very acceptably on Shakespearean subjects, and also on Tennyson. His broadcasts on Tennyson were charming and in delightful taste. He was indignant at suggestions made locally impugning the poetical gift and sense of rhythm of Adam Lindsay Gordon, and during his last illness he was busy preparing a paper on this subject.

Dr. Frank Nyulasy was loyal to the memory of his brother Arthur, who first correctly described the cardinal ligaments of the uterus, its main supports. The muscular character of these ligaments and their attachments had previously been overlooked, and it gave great satisfaction to Frank when the part played by Arthur in first describing these ligaments correctly was recognized in the writings of Victor Bonney and others.

Dr. Frank Nyulasy died on Monday, May 7, at 2 a.m. The previous evening, at about 9 o'clock, he engaged in animated conversation and attended to some professional work. For some months before his death, however, he had evidence of a failing cardiac reserve, and had from time to time symptoms of *angina pectoris*.

He leaves a widow and two sisters, all of whom are greatly attached to his memory, and to whom, in their bereavement, the sympathy of the profession will be extended.

PHILIP SYLVESTER CLARKE.

We regret to announce the death of Dr. Philip Sylvester Clarke, which occurred on June 12, 1934, at Cairns, Queensland.

Books Received.

THE DERMATERGOSES OR OCCUPATIONAL AFFECTIONS OF THE SKIN, GIVING DESCRIPTIONS OF THE TRADE PROCESSES, THE RESPONSIBLE AGENTS AND THEIR ACTIONS, by R. Prosser White, M.D., M.B., C.M., M.R.C.S.; Fourth Edition; 1934. London: H. K. Lewis and Company, Limited. Demy 8vo., pp. 732, with illustrations. Price: 35s. net.

Diary for the Month.

JUNE 26.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JUNE 27.—Victorian Branch, B.M.A.: Council.
JUNE 28.—South Australian Branch, B.M.A.: Branch.
JUNE 28.—New South Wales Branch, B.M.A.: Branch.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xiv and xv.

ROYAL NORTH SHORE HOSPITAL OF SYDNEY, NEW SOUTH WALES: Medical Superintendent.
ROYAL PRINCE ALFRED HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Officers.
THE COAST HOSPITAL, LITTLE BAY, NEW SOUTH WALES: Honorary Radiologist.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing. Lower Burdekin District Hospital, Ayr.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	Combined Friendly Societies, Clarendon and Kangarilla districts. Officer of Health, District Council of Elliston. All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

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